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Attorneys for Defendants, COUNTY OF SAN BERNARDINO and DEPUTY CHRISTOPHER ALFRED

UNITED STATES DISTRICT COURT

CENTRAL DISTRICT OF CALIFORNIA, WESTERN DIVISION

STEFFON BARBER, an individual,

Plaintiff,

v.

COUNTY OF SAN BERNARDINO, a municipal entity, and DOES 1 through 10, inclusive,

Defendant.

Case No. 5:22-cv-00625-KK-DTBx

[District Judge, Kenly Kiya Kato, Magistrate Judge, David T. Bristow]

[MOTION IN LIMINE NO. 4]

NOTICE OF MOTION AND MOTION *IN LIMINE* BY DEFENDANTS TO EXCLUDE PLAINTIFF'S EXPERT DR. BENNET OMALU [DAUBERT MOTION]; MEMORANDUM OF POINTS AND AUTHORITIES; DECLARATION OF KAYLEIGH A. ANDERSEN

Judge: Hon. Kenly Kiya Kato
Date: 1/8/2026
Time: 10:30 a.m.
Crtrm.: 3, 3rd Floor

Trial Date: 1/26/26

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TO THE HONORABLE COURT AND TO ALL PARTIES AND COUNSEL:

By and through their counsel of record in this action, defendants COUNTY OF SAN BERNARDINO and DEPUTY CHRISTOPHER ALFRED (collectively herein after as "Defendants") will move this Court for an order to exclude the following opinions and testimony of plaintiff's expert, Dr. Bennet Omalu ("Dr. Omalu"), at trial regarding:

1. Any testimony or opinions by the expert that are based on cumulative/duplicative, speculative, and/or lack foundation; and

2. Any testimony or opinions beyond the expert's scope of expertise, including all opinions and testimony regarding mental/emotional injuries, bullet trajectory, kinetic energy, and forensic incident reconstruction as to the body positioning/movement of Plaintiff on scene.

This motion is made on the grounds that the above opinions lack evidentiary support pursuant to *Daubert v. Merrell Dow Pharms*., 509 U.S. 579 (1993), and constitute nothing more than speculation. Dr. Omalu cannot state these opinions to a reasonable degree of medical probability. As such, his opinions are inadmissible under Fed. R. Evid. 702.

This motion is based on all pleadings, records and files in this action, and upon such further oral and documentary evidence as may be presented at the hearing on this motion. This motion is made following conference of counsel which took place via telephone on December 4, 2025.

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DATED: December 11, 2025

Respectfully submitted,

MANNING & KASS
ELLROD, RAMIREZ, TRESTER LLP

By: /s/ Kayleigh Andersen
Eugene R. Ramirez
Kayleigh A. Andersen
Attorneys for Defendant, COUNTY OF SAN BERNARDINO

MEMORANDUM OF POINTS AND AUTHORITIES

I. INTRODUCTION

By this motion *in limine*, defendants seek to exclude certain opinions and testimony of Plaintiff's expert, Dr. Bennet Omalu, at the time of trial, that:

1. Any testimony or opinions by the expert that are based on cumulative/duplicative, speculative, and/or lack foundation

2. Any testimony or opinions beyond the expert's scope of expertise, including all opinions and testimony regarding mental/emotional injuries, bullet trajectory, kinetic energy, and forensic incident reconstruction as to the body positioning/movement of Plaintiff on scene.

All of Dr. Omalu's opinions that relate to the above two categories should be excluded. The opinions offered by Dr. Magnusson are unreliable; they are lacking in evidentiary support and stated as speculation under the *Daubert* standard of admissibility; and are irrelevant and impossible due to Plaintiff's incarceration.

II. THE COURT SHOULD EXCLUDE DR. OMALU'S OPINIONS BECAUSE HIS OPINIONS LACK EVIDENTIARY SUPPORT AND DO NOT MEET THE THRESHOLD FOR *DAUBERT* ADMISSIBILITY.

A. The *Daubert* Threshold for Expert Opinion Evidence.

The trial court serves a gatekeeping function regarding the admissibility of expert evidence. Fed. R. Evid. Rule 702 allows admission of "scientific, technical, or other specialized knowledge" by a qualified expert if it will "assist the trier of fact to understand the evidence or to determine a fact in issue." Under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), and *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), a judge must "apply his gatekeeping role . . . to all forms of expert testimony, not just scientific testimony." *Hangarter v. Provident Life & Accident Ins. Co.*, 373 F.3d 998, 1017 (9th Cir. 2004); citing *White v. Ford Motor Co.*, 312 F.3d 998, 1007 (9th Cir. 2002).

Fed. R. Evid, Rule 702 provides that a witness may be qualified as an expert if "the testimony is based on sufficient facts or data." "Where such testimony's factual basis, data, principles, methods, or their application are called sufficiently into question . . ., the trial judge must determine whether the testimony has a 'reliable basis in the knowledge and experience of [the relevant] discipline.'" *Kumho Tire Co.*, 526 U.S. at 149. If the testimonial evidence fails in this regard, the expert opinion is inadmissible. *Id.*

In applying Rule 702, the Court functions as a gatekeeper, determining whether proffered expert testimony meets the requirements of Rule 702 by a preponderance of the evidence." *In re Countrywide Fin. Corp. Mortgage-Backed Sec. Litig.*, 984 F.Supp.2d 1021,1026 (C.D. Cal. 2013); (citing *Daubert*, 509 U.S. at 597). The offering party must show by a preponderance of the evidence that (1) the expert is qualified to render the opinion; and (2) the opinion offered has adequate factual and scientific support for that opinion. *Daubert*, 509 U.S. at 592-93.

B. Dr. Omalu Lacks The Qualifications To Render Such Opinions

Expert opinions are properly excluded if the *Daubert* standard is not met. *See Lash v. Hollis*, 2007 U.S. Dist. LEXIS 3633, *12 (E.D. Mo. 2007) (plaintiff's expert opinion on the effects of TASER ECD deployments was excluded under *Daubert* because the court found plaintiff was not qualified to testify as an expert due to his lack of familiarity with TASER devices and his reliance on only one scholarly article on the effects of TASER ECD deployments). "It is the proponent of the expert who has the burden of proving admissibility." *Lust v. Merrell Dow Pharm., Inc.*, 89 F.3d 594, 598 (9th Cir. 1996).

Dr. Omalu fails to satisfy the first prong of the admissibility analysis. He is not qualified to render a number of his trajectory opinions because he is not an expert in bullet trajectory or forensic incident reconstruction. (See Andersen Decl., Ex. A). Dr. Omalu is an Anatomic Pathologist, Clinical Pathologist, Forensic Pathologist, Neuropathologist, and an Epidemiologist, licensed to practice in California. (See

Andersen Decl., Ex. A).

Dr. Omalu does not have a specific certification that states he is certified as a bullet trajectory analyst nor has he ever authored any peer-reviewed publications where the primary topic was bullet trajectory analysis. (See Andersen Decl., Ex. A). Further, Dr. Omalu testified that he is not a certified biomechanical engineer nor does he hold any degrees or certifications in biomechanical engineering. (See Andersen Decl., Ex. A). Similarly, Dr. Omalu is not a shooting incident reconstruction expert nor does he hold any degrees or certifications in shooting incident reconstruction. (See Andersen Decl., Ex. A).

In fact, Dr. Omalu, rather than relying on any specialized knowledge and training, he simply states that bullet trajectory analysis and pattern of injuries is "common knowledge". (See Andersen Decl., Ex. A at 7).

As such, Plaintiff cannot establish that Dr. Omalu has sufficient qualifications to render his opinions regarding bullet trajectory, body position, and/or movement of the Plaintiff within the scene during the incident as it relates to the injuries Plaintiff sustained. Thus, Omalu's scientific testimony is inadmissible under *Daubert*.

C. Dr. Omalu's Opinions Lack Sufficient Evidentiary Support.

""Rule 702 requires that expert testimony relate to scientific, technical, or other specialized knowledge, which does not include unsupported speculation and subjective beliefs." *Guidroz-Brault v. Missouri Pacific R.R. Co.*, 254 F.3d 825, 829 (9th Cir. 2001); *see Daubert*, 509 U.S. at 590. "In the context of a motion for summary judgment, an expert must back up his opinion with specific facts." *Guidroz*, 254 F.3d at 831; *U.S. v. Various Slot Machines on Guam*, 658 F.2d 697, 700 (9th Cir. 1981).

Further, as of December 2023, Rule 702 will provide that "expert testimony may not be admitted unless the proponent demonstrates to the court that it is more likely than not that the proffered testimony meets the admissibility requirements set forth in the rule." *See* Fed. R. Evid. 702, Advisory Committee Notes, 2023

Amendments.

As indicated in his report identifying materials reviewed, Dr. Omalu did not personally examine any of the physical evidence that was available in this case, including discharged cartridge casings, projectiles, or other gun-related evidence collected from the scene. (See Andersen Decl., Ex. A). Dr. Omalu opines as to the velocity of the bullet and kinetic energy without citing any source for such information. (See Andersen Decl., Ex. A at 8). These opinions are outside the scope of his expertise as outlined throughout his report and CV. (See Andersen Decl., Ex. A). Dr. Omalu did not conduct any examination of Deputy Alfred's gun and cannot reasonably opine as to the velocity of a bullet from that specific gun as it relates to the kinetic energy transferred to Plaintiff's head/injuries. (See Andersen Decl., Ex. A). Dr. Omalu does not take into account the age of the gun, the weight of the gun, the weight of the bullets, the spring mechanism in the gun, or any other factors as it would relate to a calculation of kinetic energy and velocity.

Further, Dr. Omalu did not perform a scene analysis and reconstruct the incident in this case, rather Dr. Omalu relied on the "patterns of trauma in this case" that are "common knowledge" (See Andersen Decl., Ex. A at 7).

As outlined in his report, Dr. Omalu did not rely on the full spectrum of available physical, forensic, or testimonial evidence available in this case to form his opinions. As such, his opinions regarding trajectory, kinetic energy, and patterns of injury as they relate to this incident are inadmissible under *Daubert*.

D. Dr. Omalu Cannot State His Opinions To A Reasonable Degree Of Scientific Probability.

Medical expert testimony regarding causation must be stated to a reasonable degree of medical certainty. *See Lajoie v. Thompson*, 217 F.3d 663, 667.fn.4 (9th Cir. 1999. Expert testimony is not admissible if it is too wide a reach exists between the expert's opinion and the evidentiary record. *See General Elec. Co.*, 522 U.S. 136, 146 (1997) ("A court may conclude that there is simply too great an analytical gap

between the data and the opinion proferred.").

Dr. Omalu used highly speculative language throughout his report. First, Dr. Omalu cannot state to a reasonable degree of scientific probability or certainty where Deputy Alfred was located relative to Plaintiff. (See Andersen Decl., Ex. A).

Dr. Omalu conducted a superficial two-hour examination of Plaintiff at the state prison in which he is currently incarcerated. (See Andersen Decl., Ex. A at 6-7). At that time, Dr. Omalu failed to note the presence or absence of any relevant medical history indicating a possible history of head injuries (or the age at which those injuries took place) that, per Dr. Omalu, would affect the prognosis outlined in his report regarding increased potential for developing dementia or Alzheimer's. (See Andersen Decl., Ex. A at 9-19). Further, during Dr. Omalu's in-person examination, no scientific tests were conducted to evaluate or assess the neurological issues noted in the report. The conclusions reached by Dr. Omalu during his superficial examination of Plaintiff actually contradict the medical records indicating Plaintiff's motor functioning, including his ability to walk.

Dr. Oamlu's report provides a long analysis of the effects of a Traumatic Brain Injury ("TBI), without identifying the severity or extent of Plaintiff's specific TBI in this case. (See Andersen Decl., Ex. A). Dr. Omalu's report cites to a number of case studies and articles, but it is not clear whether any of these patients in the studies were suffering from a TBI as a result of a single event (like Plaintiff alleges) or from repetitive activity (akin to Dr. Omalu's work with CTE). As noted by Dr. Omalu in his report, whether the patient has had multiple or a single head injury does matter. (See Andersen Decl., Ex. A at 9-19). And, without obtaining the relevant medical history of the patient and without reviewing any previous medical records of the Plaintiff, Dr. Omalu lacks foundation to opine that the single event of a gunshot wound to the head in this case had any effect on the prognosis of Plaintiff related to an increased likelihood of dementia or Alzheimer's. Without any evidence of whether or not Plaintiff had suffered a prior head injury, the entire analysis and opinion

regarding development of certain degenerative neurologic diseases lacks foundation and the prognosis of Plaintiff cannot be stated to a reasonable degree of medical certainty.

Dr. Omalu reaches opinions about Plaintiff's pain and suffering without any explanation of the methodology in quantifying the pain and suffering. (See Andersen Decl., Ex. A at 19-24). Plaintiff is alive and can certainly testify as to his own pain and suffering. There is no scientific basis for these opinions with a living Plaintiff, and there is no scientific basis cited quantifying the subjective pain and suffering. Additionally, Dr. Omalu is not a psychologist or forensic psychologist and must be precluded from offering opinions about the mental health or emotional state of the living Plaintiff. Permitting Dr. Omalu to testify about the pain and suffering, both physical and emotional, of a living Plaintiff who was able to sit for a deposition and answer questions (and is expected to do the same at the time of trial) is irrelevant, cumulative, lacks foundation, and prejudicial to Defendants.

Dr. Omalu's conclusory testimony, replete with inability to provide an evidentiary basis for his opinions fails to meet the minimum threshold for admissibility under *Daubert*.

An expert who reaches his opinion by "cherry-picking observational studies that support his conclusion and rejecting or ignoring the great weight of the evidence that contradicts his conclusion" is not admissible because the "opinion does not reflect scientific knowledge, is not derived by the scientific method, and is not "good science." *In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1176 (N.D. Cal. 2007). Dr. Omalu is unable to state any of his opinions regarding plaintiff's prognosis to a reasonable degree of scientific probability in this matter. As such, the same constitute speculation and are inadmissible.

III. CONCLUSION

Based on the foregoing, Defendants respectfully request that the Court grant

the instant motion to exclude Dr. Bennet Omalu's opinions regarding bullet trajectory and all other unsupported and speculative scientific opinions rendered in his Rule 26(a) Expert Report and at deposition, from trial.

DATED: December 11, 2025

Respectfully submitted,

MANNING & KASS
ELLROD, RAMIREZ, TRESTER LLP

By: /s/ Kayleigh Andersen
Eugene R. Ramirez
Kayleigh A. Andersen
Attorneys for Defendant, COUNTY OF SAN BERNARDINO

DECLARATION OF KAYLEIGH ANDERSEN

I, Kayleigh Andersen, declare as follows:

1. I am an attorney at law duly authorized to practice before all the courts of the State of California and in all of the United States District Courts within the Central District of California. I am a partner in the law firm of Manning & Kass, Ellrod, Ramirez, Trester LLP, attorneys of record herein for Defendants COUNTY OF SAN BERNARDINO and CHRISTOPHER ALFRED (collectively "Defendants"). If called and sworn as a witness to testify, I am competent to testify and would testify from my own personal knowledge as to the facts set forth in this declaration, except as to those matters that are stated on information and belief herein.

2. Attached hereto as **Exhibit A**, is a true and correct copy of Plaintiff's expert Dr. Bennet Omalu's Rule 26 expert report in this matter, which was served with the Plaintiff's expert disclosures on or about October 31, 2025.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Executed on December 11, 2025 at Los Angeles, California.

/s/ Kayleigh Andersen
Kayleigh Andersen

EXHIBIT A

EXHIBIT A

Bennet Omalu
PATHOLOGY

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bennetomalu@bennetomalu.com

Autopsy and Anatomic Pathology
Clinical Pathology and Toxicology
Forensic Pathology

Neuropathology
Epidemiology
Medico-Legal Consultations

Dale K. Galipo, Esq
Law Offices of Dale K. Galipo
21800 Burbank Blvd., Suite 310
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October 31, 2025

Dear Mr. Galipo,

Re: Steffon Barber
Medico-Legal Report

Summary of Education, Training and Experience

I completed medical school in 1990 at the University of Nigeria, Enugu, Nigeria. Upon graduating from medical school, I completed a one-year clinical housemanship at the University of Nigeria Teaching Hospital in the fields of Pediatrics, Internal Medicine, General Surgery, Obstetrics, and Gynecology. After housemanship, I worked as an emergency room physician at a university hospital in Nigeria for approximately three years. I sat for and passed my United States Medical Licensing Examinations [USMLE] while I worked as an emergency room physician. I came to the United States in 1994 through a World Health Organization scholarship to become a visiting research scholar for eight months at the Department of Epidemiology, Graduate School of Public Health, University of Washington, Seattle, Washington.

In 1995, I proceeded to the College of Physicians and Surgeons of Columbia University, New York, at Harlem Hospital Center, to complete residency training in Anatomic Pathology and Clinical Pathology. In 1999, I proceeded to the University of Pittsburgh in Pittsburgh, Pennsylvania, to complete residency training in Forensic Pathology and Neuropathology. I hold four board certifications in Anatomic Pathology, Clinical Pathology, Forensic Pathology and Neuropathology. I also hold a Masters in Public Health [MPH] in Epidemiology from the Graduate School of Public Health at the University of Pittsburgh in Pittsburgh, Pennsylvania. I also hold a Masters in Business Administration [MBA] degree from the Tepper School of Business at Carnegie Mellon University in Pittsburgh, Pennsylvania, one of the leading business schools in the world. I am a Certified Physician Executive and an Honorary Fellow of the American Association of Physician Leadership [AAPL]. I also hold a fifth board certification in Medical Management from the AAPL. I am currently licensed to practice Medicine and Surgery in the State of California.

I am currently the President and Medical Director of Bennet Omalu Pathology [BOP], a California medico-legal consulting firm, and a Clinical Professor at the Department of Medical Pathology and Laboratory Medicine, University of California, Davis. In my capacity as the

Medical Director of BOP, I am a consulting Forensic Pathologist and Neuropathologist to many hospitals in central California and to several counties in northern California. There are less than a few dozen practicing Forensic Pathologists-Neuropathologists in the United States who are board-certified in both Forensic Pathology and Neuropathology.

For over 25 years, I have been involved in over 15,000 death and injury investigations in my career as a Forensic Pathologist and Neuropathologist, which began in 1999. I have personally conducted and performed over 13,000 autopsies and death investigations and examined over 15,000 brain tissue specimens. I also perform trauma pattern analysis in both living patients and deceased patients to determine causes and mechanisms of sustenance of injuries and death. I am also involved in the evaluation of living victims of all types of injuries and trauma, including, but not limited to, victims of assault, traumatic falls, industrial and accidental injuries, medical complications and misadventures, rape, child abuse, and sports-related injuries.

I have performed autopsies and examined the medical records, occupational histories, exposure histories, autopsy tissues, and biopsy tissues of hundreds of living and deceased patients who had been occupationally exposed to asbestos, solvents, and other types of toxic agents. I have performed differential diagnosis, made disease diagnosis, and determined medical causation of diseases involving all types of occupational exposures, including asbestos-related diseases and malignancies like malignant mesothelioma.

I have been consulted and retained as an expert witness in 2,000-3,000 cases involving all types of medico-legal cases across all jurisdictions in the United States, including federal, state, county, and municipal courts and arbitration panels, in both civil and criminal cases, for the plaintiff, defense, district attorneys, and public defenders. I have been involved as an expert witness in complex class action and industrial lawsuits involving thousands of individuals and major corporations.

My areas of interest and focus include brain pathophysiology, brain injuries, and brain trauma, in both living and deceased patients. I identified Chronic Traumatic Encephalopathy [CTE] in a retired football player when I performed an autopsy and examined the brain of Mike Webster in 2002. Subsequently, I identified CTE in other high-impact, high-contact sports athletes and in military veterans suffering from Post-Traumatic Stress Disorder [PTSD]. Since 2002, CTE has received international attention from the sports industry, sports medicine, and neuroscience. My work has been featured extensively in all media platforms across the world. My work and life were featured in a major Hollywood film, *Concussion*, released in December 2015 by Sony Motion Pictures, in which the renowned actor, Will Smith, played me as Dr. Omalu. Several New York Times best-selling books have also been published on my life and work, including *The League of Denial* and *Concussion*. I have published several books including my memoir, *Truth Doesn't Have a Side*, which was published in August 2017. My latest book, *Brain Damage in Contact Sports,* was published in February 2018. I have published extensively in the medical and scientific literature, authoring many scientific papers and book chapters.

I have received three honorary PhD degrees from two universities in the United States and from the Royal College of Surgeons of Ireland in recognition of my work and expertise. I have also received numerous awards from across the world in recognition of my work and expertise in both living and deceased patients. I have received the "Distinguished Service Award" from the American Medical Association [AMA], which is the most prestigious award of the AMA. I have

been honored by the United States Congress, and I have appeared on multiple occasions before committees of the United States Congress and committees of State Legislatures across the Unites States, advising them on matters relating to trauma. In 2019 and 2020, I was appointed to the Traumatic Brain Injury Board of the State of California to advise the state on matters relating to traumatic brain injuries.

Since 1999, I have testified as an expert witness in matters relating to all types of injuries and deaths in over 800 court proceedings across the United States. I have attached a copy of my curriculum vitae, which enumerates my body of work and experience in greater detail. The cases I have testified in, beginning in 2009, are enumerated at the end of my curriculum vitae.

Pursuant upon your request, I have reviewed the following materials sent to me on the case of Steffon Barber, Deceased:

1. State of California, County of San Bernardino search warrant and affidavit for Arrowhead Regional Medical Center to obtain hospital admission blood, copies of medical records, doctor's reports, X-Rays and lab results for patient Steffon Barber.
2. Medical records obtained from Arrowhead Regional Medical Center pursuant to the search warrant.
3. Medical records from San Bernardino County Sheriff's Department.
4. Second Amended Complaint.
5. Deposition of Deputy Alfred.
6. Deposition of Steffon Barber.
7. Audio belt recording of Deputy Alfred.

In order to perform and apply a valid differential diagnosis method including but not limited to causation criteria analysis[1], Central Limit Theorem analysis and Clinico-Pathologic Correlation analysis, on this case, I had to review, document, and analyze the materials sent to me on this case in considerable depth and detail. I also visited the patient, Steffon Barber at the Salinas Valley State Prison, spent two hours, interacted with him, and performed a systems review on him. Such differential diagnosis and reviews would form the foundation for my case-specific and general causation opinions in this case.

Brief Summary of the Prevailing Forensic Scenario[1]

Steffon Barber was born on February 12, 1986. On April 27, 2021, at the age of 35 years, he sustained severe traumatic brain injury [TBI] when he was shot in the head by a police officer.

Medical Records from Arrowhead Regional Medical Center
04/28/2021
Steffon Barber was brought in by air transport to the emergency room at 12:22 a.m. with a history of a severe TBI from a gunshot wound to the posterior vertex of the head and parietal-occipital scalp, with cervical spine tenderness. He had a respiratory rate of 16/min with an oxygen saturation of 100 % in room air. He was oriented in person, and had a Glasgow Coma

[1] This section of the report should not be used to establish the facts in this case and is not intended to be used to establish the facts in this case.

Score (GCS) of 13. There was a 3 cm full-thickness laceration on the right side of the vertex with exposed brain tissue. The cervical spine was tender to palpation.

Orbital-facial computed tomography (CT) showed no significant orbital, facial, or mandibular fractures. There were normal sinuses. Cervical spine CT revealed no acute compression fracture or subluxation from C1 through T3.

CT of the head showed the following:
"Findings: There is comminuted fracture of the right parietal skull with extensive bullet and bone fragments tracking centrally into the brain. There is mild bilateral subarachnoid hemorrhage toward the vertex, subdural hemorrhage along the falx and right tentorium. There is no midline shift. Basal cisterns are still visualized. The visualized paranasal sinuses and mastoid air cells are clear. There are no erosive changes in the skull base. The remaining sulci and ventricles are within normal limits. The remaining cerebral parenchyma, brainstem and cerebellum appear to be normal. The superior sagittal and transverse sinuses are within normal limits. There are no definite findings to suggest hyperdense sign in the bilateral middle cerebral and basilar arteries. The ocular globes are intact and symmetric bilaterally.

Impression: Gunshot wound to the right parietal vertex with comminuted fracture and bullet/bone fragments tracking centrally into the brain. Bilateral parietal subarachnoid hemorrhage. Subdural hemorrhage along the falx and right tentorium. No gross herniation at this time."

A computed tomography angiography (CTA)/computed tomography venography (CTV) showed no apparent vascular injury or aneurysm/sinus injury. Electrocardiogram (ECG) revealed sinus bradycardia with marked sinus arrhythmia. There was early repolarization (ST elevation with normally reflected T-wave).

During a neurosurgical review, it was noted that Steffon Barber was confused but was able to move his four extremities. There was an open wound at the right parietal vertex, three centimeters off the midline, with the herniation of brain matter and a blood clot. There was no active bleeding. He was intubated in the trauma bay and continued to remain hemodynamically stable.

The primary impressions were gunshot wound of the head, traumatic encephalopathy and traumatic subarachnoid hemorrhages.

Mr. Barber was subsequently admitted to the surgical intensive care unit (SICU) and was commenced on empirical antibiotics. He was prescribed and administered Keppra and Dilantin for seizure prophylaxis. Continuous elevation of the head of the bed to 30 degrees was recommended.

He underwent a bedside washout, irrigation and debridement of a right open gunshot wound with depressed skull fracture, dural defect and extruding brain. There were necrotic brain tissues and bone fragments with dark blood clotting in an open gunshot wound in the vertex of the right parietal scalp. He also underwent titanium cranioplasty and subgaleal drain placement. There were a 3 cm in length wound with jagged edges, disrupted and frayed galeal edges, comminuted skull fractures, herniating liquified brain tissue and hematoma from the cranial defect, and bleeding from the brain tissue.

04/29/2021
Mr. Barber remained intubated with a GCS of 11T. His postoperative CT showed no new hemorrhage. He followed simple commands in the right upper extremity, such as thumbs up. There was no movement in the left upper extremity or lower extremity. He could withdraw in the right lower extremity. The surgical repair site was covered with a clean and dry dressing. Dr. Li advised a plan for extubation and the use of sequential compression devices (SCDs) for prophylaxis. Physical therapy (PT), occupational therapy (OT), and speech therapy (SP) consults were ordered.

04/30/2021
Mr. Barber's GCS improved to 13. All subgaleal drains were removed, and a staple was placed at the drain exit site. The procedure was well-tolerated, and staple removal was scheduled for the 14th postoperative day.

05/01/2021
Mr. Barber was transferred to the internal medicine team for the continued medical management of his traumatic brain injury. His surgical diagnoses remained a gunshot wound to the head, traumatic subarachnoid hemorrhage, and traumatic encephalopathy.

05/03/2021
Dr. Schiraldi evaluated Mr. Barber and recommended a diagnostic angiogram to identify any potential pseudoaneurysm and address it to prevent future hemorrhage. The benefits, risks, and alternatives were discussed, and Mr. Barber expressed understanding and consented to proceed with the procedure. He was also deemed medically stable for transfer to the Moreno Valley Riverside University Health System (RUHS) Jail ward, and Dilantin therapy was discontinued.

05/04/2021
Mr. Barber was planned for a digital subtraction angiography (DSA).

05/05/2021
The planned DSA was rescheduled for Friday. Mr. Barber's transfer to the Moreno Valley RUHS Jail ward was to take place following completion of the procedure.

05/6/2021
Mr. Barber underwent a diagnostic cerebral angiogram via right femoral artery access, performed by Dr. Schiraldi, which revealed no significant cerebrovascular abnormalities. A physical therapist evaluated him and recommended acute rehabilitation, as he required maximum assistance due to his neurological deficits. Owing to his condition, he was not yet fit to return to jail but was deemed stable for transfer to the TSS team. He was assessed as not requiring antiepileptic medications or antibiotics. Staple removal was scheduled for May 12, 2021, and the right groin bandage was to be removed on May 8, 2021.

05/10/2021 – 5/11/2021
Mr. Barber exhibited generalized weakness and lethargy and was advised to increase the frequency of his physical therapy sessions.

05/12/2021
Mr. Barber reported functional improvement with physical and occupational therapy. He demonstrated increased movement in his right lower extremity and limited motion on the left

side of his body, though he was able to improve mobility during therapy sessions. Sensation was decreased in the left upper extremity (LUE) and both lower extremities (LE). Neurosurgery was consulted for a wound check evaluation.

05/13/2021
Mr. Barber had his staples and drain removed, and the scalp surgical incision was noted to have healed well.

05/14/2021
Mr. Barber had muscle spasms in his left leg overnight and was given oral Robaxin.

05/19/2021
Mr. Barber attended a court hearing and reported that he had slid off his chair but did not experience a fall. He complained of buttock pain, which he attributed to sitting in a wheelchair for an extended period.

05/21/2021
Mr. Barber reported having a headache but declined medication. His symptoms resolved after elevating the head of his bed by 30 degrees.

Records from the San Bernardino County Sheriff-Coroner Department indicate the following diagnoses for Steffon Barber stamped 10/08/2021:
1. Hemiplegia and hemiparesis
2. Personal history of trauma brain injury

Dr. Bennet Omalu's visit and clerkship of Steffon Barber at the Salinas Valley State Prison on October 29, 2025[2]
I visited Steffon Barber at the Salinas Valley State Prison on October 29, 2025. I met with him for two hours beginning at 08:20 a.m. and ending at 10:20 a.m. I performed clinical clerkship and systems review of him by interviewing him and examining him physically. I reviewed his family, social and developmental history.

He was wheelchair bound and could not ambulate independently without the wheelchair. He had significant loss of power and weakness of his upper and lower extremities, accentuated on the left side. He appeared very reserved and subdued, drained and tired. He spoke very softly and became tearful intermittently.

After the shooting, Steffon Barber attempted to get out of bed at the hospital and a nurse told him not to do it, that he was paralyzed and could not walk. His entire left side was paralyzed. He was told that he was shot in the head by a police officer. At this time, he showed me his head, with two large scars on the vertex of the head in the bilateral posterior frontal and parietal scalp and the right temporal scalp.

He was wounded badly and until this day, his left upper and lower extremities were "messed up badly". He could not walk and is dependent on his wheelchair for ambulation.

[2] My interactions and conversations with Steffon Barber were not recorded and could not be recorded given that I met him in a prison setting as an incarcerated inmate.

Steffon stated that he felt disabled. “Everything is so difficult to do”. He experienced constant pain all over his body especially constant stinging and needle-pricking pain in his lower extremities, which never goes away. He has good days and bad days.

He suffered intermittent headaches and has noticed that he was becoming more and more forgetful. He can comprehend stuff, but retaining things and what he has read is becoming a lot harder and difficult. He stated that he felt depressed and humbled. “I am disabled, I cannot do things I used to do, and will like to do. I will like to run. I will like to shoot a hoop or two from time to time. But no, I can no longer do any of that. I am disabled.”

Steffon had wanted to be an underwater welder. He could not swim and was planning to learn how to swim. But now, he cannot do any of that. He cannot learn how to swim and has to forget his dreams of being an underwater welder. “That is what I have always wanted to do”.

Medico-Legal Questions

1. **What were the characteristics and trajectory of the bullet of the gunshot wound Steffon Barber sustained?**
    a. **What was Steffon Barber’s body positioning while he was shot?**
    b. **What injuries or damages were caused to Steffon Barber by the gunshot?**

Medicine is a life science, which is evidence based. The practice of medicine is guided by established standards and generally accepted principles, which certified physicians must adhere to. The specialties and the categories of physicians who are most proficiently trained, specialized, and competent in the accurate determination of the cause, mechanism and manner of death and the mechanisms of sustenance of serious bodily injury and possibly lethal trauma are the forensic pathologists. Steffon Barber suffered serious bodily injuries.

It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of traumas and injuries. Applying the clinico-pathologic method of differential diagnosis, a specific documented pattern of trauma can be evaluated, translated, and applied to the determination of the mechanisms of generation, causation, and sustenance of the specified trauma pattern, with a reasonable degree of medical and scientific certainty; based on the differential diagnosis of the established common knowledge and generally accepted principles of trauma patterns and their mechanisms of generation, causation, and sustenance. The documentation and translation of patterns of trauma can be done by a variety of methods including but not limited to radiological methods, autopsy methods, clinical physical examination methods, and applied clinico-pathologic methods.

The patterns of injuries generated by gunshots, firearms and ballistics weapons, and the mechanisms of generation, causation, and sustenance of these patterns of injuries are very well-established in the medical literature and are common knowledge. Based on the prevailing forensic scenario, and on the generally accepted principles and common knowledge of medicine and science, and based on the global constellation, configurations and anatomic conformations

of the gunshot wound sustained by Steffon Barber, the mechanisms of generation, causation and sustenance of his injuries can be determined with a reasonable degree of medical certainty.

Based on the physical characteristics and physics of ballistics, partially burnt and hot residues of gunpowder and soot travel behind the bullet when it exits the muzzle, and due to gravitational forces and the differential densities of the bullet, soot, and residues of gunpowder in the gravitational field, the bullet can travel longest, followed by the partially burnt gunpowder residues, which travel longer than soot. Soot will travel for about 1 foot, before it is pulled down by gravitational forces, and the partially burnt gunpowder residue will travel for about 2-3 feet before it is pulled down by gravitational forces. Therefore, if the muzzle of the gun were closer to the skin by less than 1 foot, you would expect to find marginal soot deposits around the gunshot wound of entrance [close range shot]. If the muzzle of the gun were closer to the skin by less than 2-3 feet, you would expect to find powder stippling around the gunshot wound of entrance [intermediate range shot]. If the muzzle of the gun were located greater than 2-3 feet away from the skin ad infinitum, you would expect to find only marginal abrasions around the wound without soot deposits or powder stippling [distant range shot]. If there is an eccentric accentuation of the width of the marginal abrasion, it may suggest that the muzzle of the gun was not located perpendicularly to the skin when it was fired but rather located in the direction of the eccentric accentuation of the marginal abrasion.

The direction of travel of a bullet inside the body can be determined in the three planes of nature with the body disposed in the universal anatomic position, by the systematic tracking and description of the anatomic pathway of the bullet, tissue disruptions, damages and injuries, correlated with the anatomic topographic locations of the gunshot wound of entrance, gunshot wound of exit and recovery of the bullet, or where the bullet settled.

Based on these common knowledge and generally accepted principles of medicine and science, Steffon Barber sustained a gunshot wound of his head, with the entrance wound located in the vertex of the head in the right medial and posterior frontal and parietal scalp. The anatomic location of the gunshot wound of entrance indicates that at the time he was shot, Steffon Barber's head was located at a vertical level that was lower than the vertical level of the gun that fired the bullet. Steffon Barber was not standing erect on his feet when he was shot. The prevailing forensic scenario suggests that Steffon Barber was sitting down in a car when he was shot. This scenario would be consistent with the anatomic location of the gunshot wound of entrance in the vertex of the head.

The bullet perforated, contused and lacerated the dorsal right posterior frontal and parietal scalp, perforated and fractured the frontal and parietal calvarium, perforated, contused and lacerated the dorsal dura mater and meninges, perforated, contused and lacerated the right cerebral hemisphere where the bullet fragments came to settle. There were extensive contusions and lacerations of the right dorsal cerebral hemisphere with parenchymal pulpifaction, subdural and subarachnoidal hemorrhages. Steffon Barber suffered severe traumatic brain injury caused by the gunshot wound and bullet.

He suffered both focal and diffuse, primary and secondary severe traumatic brain injury with a cavitatory effect given that the cranial cavity is an enclosed space, and a bullet penetrates this enclosed space at very high velocities of greater than 1200 feet per second, and transfers large amounts of kinetic energy into the intra-cranial cavity and brain.

The bullet traveled in a forward, downward and rightward trajectory into the brain. The anatomic locations, configurations and conformations of the gunshot wound are consistent with the shooter and gun located at back of Steffon Barber's head when the bullet was fired. Steffon Barber was not facing the officer who shot him when the bullet was fired.

The clinical documentations and descriptions of the gunshot wound of entrance on Steffon Barber's head did not mention any soot deposits or powder stippling. This would mean that Steffon Barber suffered a distant shot. He was not close to the officer who fired the bullet. He was located at a distance that was far removed from the muzzle of the gun, at greater than 2-3 feet ad infinitum. The gunshot wound Steffon Barber suffered was not a contact gunshot wound, it was not a loose contact or close range gunshot wound. It was not an intermediate range gunshot wound. It was a distant range gunshot wound.

The global configurations and conformations of the gunshot wound of the head that Steffon Barber sustained are consistent with the stated scenario in Steffon Barber's deposition that Steffon Barber did not see the officer who shot him, he did not know that it was an officer and had his back facing the officer when he was shot. He was not charging at the officer with his car or attempting to assault the officer with his car. He was not facing the officer who shot him, when he was shot. He was sitting in the seat of a car when he was shot by an officer who was standing on his feet. The vertical level or height of the muzzle of the gun was either higher or about the same vertical height as the level of his head when the bullet was fired. The bullet entered the head from the top of the head, and in the back of the head.

2. On April 27, 2021, Steffon Barber sustained severe traumatic brain injury when he was shot in the head by a police officer. He was diagnosed with a gunshot wound of the head and traumatic encephalopathy at the hospital.
   a. Did the severe TBI cause permanent brain damage?
   b. What are the expected long term effects, outcomes and sequelae of his severe TBI?
   c. Does the severe TBI he suffered independently, significantly and substantially increase his risk of developing neurodegenerative diseases including but not limited to all types of dementias, traumatic encephalopathy and chronic cerebrovascular disease?

As has been presented above, yes, Steffon Barber suffered severe TBI on April 27, 2021 when he was shot in the head by a police officer. The severe TBI he sustained from a gunshot wound of his head is an independent, significant and substantial contributory, aggravating and accelerating factor for neurodegenerative diseases including but not limited to dementias, traumatic encephalopathy and chronic cerebrovascular diseases.

The differential diagnosis of his medical history, which has been summarized above confirms that the severe TBI and gunshot wound of the head he was diagnosed with caused substantial permanent, progressive and cumulative brain damage and serious bodily injury that is consistent with traumatic encephalopathy, a neurodegenerative disease and a type of dementia, which will progress as time goes on and as he gets older into more advanced forms of dementias and neurodegenerative diseases including Alzheimer's Disease pathological changes in the brain

All forms of chronic brain damage and neurodegeneration following TBI, traumatic encephalopathy and all forms of dementia belong to one and the same spectrum of diseases or dementias[2].

The human brain is a post-mitotic organ and as a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself following injury and trauma-induced cellular damage. This means that when the human brain suffers any type of irreversible injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy, and as time progresses the permanent brain injury may progress into a neurodegenerative disease. All forms of traumatic injuries to the brain, including the milder forms of TBI like a concussion are permanent brain injuries which can be progressive with time[3].

A history of exposure to single, episodic, or repetitive TBI is reliable differential diagnosis evidence for the reasonable determination of permanent brain damage and the expected outcomes and sequelae of TBI. This means that physicians reasonably rely on the patient's medical, social, educational, and occupational histories as narrated by the patient, family members, friends and colleagues to perform this differential diagnosis. A calculation of the quantitative threshold dose of exposure to TBI has never been required for the valid differential diagnosis of TBI outcomes and sequelae, and to link a patient's TBI exposure to these outcomes and sequelae. It is an accepted methodology across health organizations and specialties to use exposure history for the differential diagnosis of injury outcomes across all diseases and disease syndromes. This is an established standard of practice.

Medicine is a life science, which is evidence based. The practice of medicine is guided by established standards and generally accepted principles, which certified physicians must adhere to. Differential Diagnosis and trauma pattern recognition, interpretation and analyses are the fundamental methodologies physicians adopt in the determination of expected and anticipated outcomes and sequelae of disease and trauma. It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of injuries, outcomes, and sequalae.

After I described traumatic encephalopathy in the brains of football players in 2002 in Pittsburgh, Pennsylvania I performed and applied the method of differential diagnosis and the Bradford Hill Criteria[1,4] to determine the contributory and causal risk factors for TBI and traumatic encephalopathy. Traumatic encephalopathy was defined as the "disturbance of structure and/or function of nerve cells, glia, or intracranial vessels resulting from injury"[5]. Such a disturbance is expected to manifest with a constellation of syndromic signs and symptoms with time and over time following traumatic brain injury. I performed an extensive review of the medical literature for traumatic brain injury and traumatic encephalopathy beginning with Hippocrates who first recognized and named concussions at about 400 B.C.[6,7], followed by Claudius Galenus in the 2nd century AD, who both named concussions commotio cerebri[6,7].

In my continuing differential diagnosis review and method, which has spanned across the centuries from about 400 B.C. to 2022, I was able to determine that traumatic encephalopathy was not a novel or new disease, and that traumatic encephalopathy has been a disease that was very well-recognized and accepted by doctors across the world for centuries[7]. The disease traumatic encephalopathy had been known by a variety of names across the centuries, one and same disease, but different labels, syntax and names, given by different doctors, researchers and agencies[7,8] across the world and global scientific community. In 2014, I published a paper and

book chapter titled "Chronic Traumatic Encephalopathy"[8] in which I listed the names I had identified that traumatic encephalopathy had been known by over the centuries. The table is presented below, copied from page 40 of the article:

Table 1. Syntactics and semantics of CTE across the centuries

1. Cerebral neurasthenia
2. Chronic postconcussion syndrome
3. Chronic TBI/chronic brain injury
4. Compensation hysteria
5. Concussion neurosis
6. Delayed traumatic apoplexy
7. Dementia pugilistica
8. Dementia traumatica
9. Encephalopathia traumatica
10. Litigation neurosis
11. Postconcussion neurosis
12. Postconcussion syndrome
13. Posttraumatic concussion state
14. Posttraumatic dementia
15. Posttraumatic head syndrome
16. Posttraumatic parkinsonism
17. Posttraumatic psychoneurosis
18. Posttraumatic stress disorder
19. Punch drunk/punch-drunk state
20. Terror neurosis
21. Traumatic constitution
22. Traumatic encephalitis
23. Traumatic encephalopathy
24. Traumatic encephalopathy of boxers
25. Traumatic hysterias
26. Traumatic insanity
27. Traumatic neurosis
28. Traumatic psychosis

In 1964, the United States Congress of Neurological Surgeons formed an Ad Hoc Committee to study head injury nomenclature, and their report was published in 1966 titled *Proceedings of the Congress of Neurological Surgeons in 1964: Report of the Ad Hoc Committee to Study Head Injury Nomenclature*[5]. In this report United States neurosurgeons identified and described traumatic encephalopathy as "Disturbance of structure and/or function of nerve cells, glia, or intracranial vessels resulting from injury."[5]

The differential diagnosis method is a very well established and generally accepted methodology in the medical sciences and is common knowledge for the determination of disease outcomes. The generally accepted principles and standards of practice of the differential diagnosis of TBI and the sequelae of TBI are based on the qualitative/quantitative history of exposure to TBI like Steffon Barber was exposed to on April 27, 2021.

The type of gunshot wounds that Steffon Barber suffered April 27, 2021 are commonly known to cause TBI and permanent brain damage[9-14]. Such a violent high-velocity injury causes the transfer of linear/ translational and angular/ rotational acceleration-deceleration forces and kinetic energy to the skull and brain, which is known to cause primary and secondary TBI with cerebral edema, brain swelling and herniation, cerebral hypoperfusion and raised intracranial pressure, axonal shearing, focal and diffuse traumatic axonal and micro-vascular brain injury, neuropil contusional hemorrhages and necrosis, parenchymal lacerations and permanent brain damage[11-17].

In this case of Steffon Barber, he began manifesting the symptoms and signs of TBI immediately after he was shot. He manifested, continues to manifest, and is suffering from the symptoms and signs of TBI, TBI outcomes and sequelae, neurodegenerative disease and dementia, as have been clearly demonstrated by his prevailing medical history, which has been summarized above. In addition to the signs and symptoms of acute mild, moderate and severe TBI, exposure to TBI may present immediately or after a delayed interval with post-concussion syndrome, post-traumatic epilepsy, post-traumatic encephalopathy [PTE], mood disorders, behavioral disorders, mild cognitive impairment, neuropsychiatric disorders including drug abuse and alcoholism, motor disorders including but not limited to cerebellar ataxia, motor neuron disease and Parkinson's disease, dementia including Alzheimer's Disease, CTE, Post-Traumatic Stress Disorder, neurovascular diseases, somatic and neurosensory symptoms like headaches, body aches and pain, and increased risk of suicide[8,18-71]

While TBI has been confirmed to be a causal risk factor for developing various dementias and neurodegenerative diseases as has been stated above, dementias like Lewy Body Disease/ Dementia [Parkinson's Disease] have exhibited one of the strongest and most compelling link to TBI as a causal risk factor amongst other spectrum of dementias and neurodegenerative diseases[25,49,52-57].

For a patient like Steffon Barber, the expected outcomes of a disease or injury can be further analyzed and assessed epidemiologically using published epidemiological studies founded upon previously established and generally accepted principles of medicine and science. As an epidemiologist, I would apply many published long-term population-based epidemiological studies to the case analysis of Steffon Barber's expected TBI risk outcomes and sequelae.

Exposure to all forms of TBI is associated with elevated risks of impaired adult functioning across all outcome measures[39] in a patient like Steffon Barber. Exposure to traumatic brain injury may be associated with and is an independent risk factor for premature deaths at younger ages, with approximately 24.3 years of life lost[38,39,72,73] and unadjusted odds ratio of 3.6 for premature death among traumatic brain injury patients, with increased risks for all causes of premature deaths, and even higher absolute rates of death in patients with co-mobidities[38,39].

There is a 400-fold increased life-time diagnosis of substance abuse in patients with a history of traumatic brain injury[38,39]. There is a dose-response relationship with outcome measures and injury severity with recurrent or repetitive traumatic brain injury associated with higher risks sometimes up to 3-fold increased risk compared to single-episode traumatic brain injury[38,39] observed with injury severity.

The human brain is a post-mitotic organ and as a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself following injury and trauma-induced cellular damage. This means that when the human brain suffers any type of significant or irreversible

injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy, and as time progresses the permanent brain injury may progress into a neurodegenerative disease.
Mild TBIs and concussions can aggravate dementia in a patient already suffering from dementia. Even a single episode of a mild TBI or a concussion is associated with an increased risk of cognitive decline and dementia. This association is, however, more pronounced with more severe TBIs or repeated mild TBIs[24,74]. A history of TBI may cause cognitive impairment to appear two or more years earlier than it would otherwise, and is a substantial risk factor for cognitive decline in older adults with the beginning of moderate cognitive impairment and Alzheimer's Dementia[75].

In a nationwide population-based observational cohort study in Denmark, citizens data from national registries of all people born in Denmark who were living in the country on January 1, 1995, and who were at least 50 years old at some point during follow-up, 1999-2013, were studied. Information on TBI was obtained from the Danish National Patient Register [NPR]. Information on dementia was obtained from by combining data recorded in the NPR, the Danish Psychiatric Central Register, and the Danish National Prescription Registry. The long term risk of dementia after TBI was established using survival analysis. Data for a cohort of 2.8 million people were used for a total of 28 million person years at risk of dementia. 132 thousand individuals [4.7%] had at least one TBI, and 127 thousand [4.5%] had incident dementia during the study period. The risk of dementia was highest within the first six months after TBI, and also increased with increasing number of events. TBI was associated with an increased risk of dementia both compared with people without a history of TBI and people with non-TBI trauma. The overall risk of dementia in individuals with a history of TBI was 24 percent higher than those without a history of TBI. A single episode of severe TBI increased the risk by 35 percent. A single episode of mild TBI or concussion increased the risk by 17 percent. Even a single episode of mild TBI was associated with a significantly higher risk of dementia[24].

"Dementia-related manifestations after TBI include abnormalities of memory, thinking and concentration, communication, interactions with others, mood, and personality. TBI may cause rapid, complex structural, and physiological changes in the brain, that in addition to the released biomarkers, subsequently lead to an abrupt coping crisis and abnormal responses like excessive anxiety and depression. This disorder might happen when symptoms from psychological trauma disrupt daily functioning for at least a month. A study showed that during this time, subjects who sustained TBI were 4–6 times as likely to develop dementia than those without TBI. Furthermore, a concussion (mild TBI) or other TBI can increase the risk of developing dementia even after 30 years of the primary insult"[76].

"There was a significant interaction between TBI severity and age category such that moderate/severe TBI was associated with increased dementia risk across all ages, while mild TBI became a relatively more important dementia predictor with increasing age" in the elderly[77].

"Overall, TBI has been reported to confer a 1.6- to 3.7-fold increased risk of dementia… Furthermore, TBI may impact cognitive function to a different degree depending on the age of onset. Previous research has suggested that slow cognitive decline may occur after a TBI at any age though this decline appears to be more severe among individuals who experience a TBI at an older versus younger age. This may be related to brain plasticity. Older individuals may have less ability to compensate for TBI-related brain damage during the initial recovery period or may experience greater brain degeneration after the initial recovery period due to reduced plasticity of the aging brain. Therefore, when exploring the relationship between TBI and dementia, the timing

of TBI over the life course deserves more attention...[78] The odds ratio of dementia was 1.27 for TBI at any age, 1.55 for TBI at 50 to 59 years, and 1.67 for TBI at 60 to 69 years. TBI across all ages including the elderly is associated with an increased risk of dementia[78].
Steffon Barber exhibited novel and progressive signs and symptoms of TBI and permanent brain damage following his April 27, 2021 gunshot wound of the head. In an epidemiological population-based study by Nordstrom A and Nordstrom P, it was confirmed that the risk of a dementia or neurocognitive disorder diagnosis following TBI was highest during the first year after the TBI[79]. In this study all individuals aged 50 years old and older on December 31, 2005, were included for a total of 3.3 million persons. The diagnosis of dementia and TBI were tracked through nationwide databases for seven years until December 31, 2012. Individuals diagnosed with TBI were matched with up to two controls, and individuals diagnosed with dementia were matched with up to two controls. The mean follow-up period was 15.3 years. 6.3% of the participants with TBI were diagnosed with dementia with an adjusted odds ratio of 1.81. The association between TBI and dementia was strongest in the first year after TBI, but the risk remained significantly greater than 30 years after TBI. The history of TBI may accelerate the age of onset of cognitive impairment by two or more years[75].

The case of Steffon Barber is not an outlier and is not an anomaly. He was a representative index of TBI in human beings, as a member of the human race. The differential diagnosis method identifies and confirms that exposure to TBI especially blunt force trauma of the head in any form of human activity is the single most important risk factor for traumatic encephalopathy and dementia[7]. Over 2000 years after the work of Hippocrates in identifying concussions, Ling H et al. at the Institute of Neurology, University of London have reported that about 12% of the general population suffer from traumatic encephalopathy due to exposure to TBI[80]. Steffon Barber is one of these 12%.

A young patient like Steffon Barber who sustained severe TBI at the age of 35-years-old, more likely than not, possesses a significantly and substantially increased risk of developing Young-Onset Dementia [YOD], which is defined by the onset of dementia before the age of 65-years-old[81]. There is a strong association between YOD of non-AD forms and TBIs of different severity. In a national wide cohort study Nordstrom P et al[81] studied a cohort of about 810,000 Swedish men with a mean age of 18 years old, who were conscripted for military service between 1969 and 1986. TBIs, dementias and covariates were extracted from national registers. Time-dependent exposures were evaluated using Cox proportional hazard regression models. There was a median follow-up of 33 years. One severe TBI like we have in this case was associated with a high hazard ratio of 11.4 for YOD of non-AD forms[81].

In their paper titled “Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias”, Plassman BL et al[82] reported their findings in a population-based prospective historical cohort study. The study included men who were world war II navy and marine veterans in 1944 to 1945 who were hospitalized during their military service with a diagnosis of either a non-penetrating head injury or another unrelated condition. The entire sample was then evaluated in 1996 to 1997 for dementia and AD using a multistage procedure. There were 548 veterans with head injury and 1228 veterans without head injury who completed all assigned stages of the study. Both moderate and severe TBI were associated with significantly increased risks of dementia and AD with hazard rations of 2.32 and 4.5, respectively. They concluded that moderate and severe head injuries in early adult life were associated with increased risk of AD and other dementias in late life. The risk of dementia and AD increased with the severity of the TBI[82].

Using the Swedish Twin Registry, over 35,000 dementia-free twins were followed up for up to 18 years and TBI history was identified using medical records. In a multi-adjusted generalized estimating equation model, there was an odds ratio of 1.03 to 1.57 for developing dementia following TBI at any age; and odds ratio of 1.12 to 2.49 for TBI at 50-59 years old, and an odds ratio of 1.12 to 2.49 for TBI at 60-69 years old. Zhang L et al[78] concluded that TBI at any age but especially between the ages of 50-69 years old, was associated with significantly and substantially increased risks of developing dementia[78].

In yet another nationwide population based study, Yang JR et al[34] used Taiwan's National health Insurance Research Database to identify 501,889 adults who had ≥1 medical record of craniofacial trauma between 2000 and 2010 and did not have a dementia diagnosis at baseline. Diagnosis of craniofacial trauma including facial bone fracture and TBI, and dementia were made using ICD-9 codes. The standardized incidence ratio was used to determine whether craniofacial trauma was associated with a greater risk of incident dementia compared with the general population. The Cox proportional hazards model was used to predict the risk of dementia among the trauma cohort by comparing patients with and without comorbidities. Out of 501,889 patient with craniofacial trauma, 7804 [1.5%] developed dementia. Facial bone fracture was shown to be associated with an increased dementia risk compared with the general population. Craniofacial trauma accompanied with postinjury comorbidities was associated with an increased risk of dementia during follow-up periods compared with the group without comorbidities. Yang JR et al concluded that craniofacial traumas were significantly and substantially associated with an increased risk of subsequent dementia[34].

In order to assess the association between TBI and dementia, Simmonds, E et al[83] performed a population-based study using Welsh [UK] electronic health records, which included 20 years of data and 1.7 million individuals with hospital or general practitioner diagnoses of dementia and TBI. Study participants were between the ages of 30 and 65 years old in 1999 without a previous dementia diagnosis. The long-term risk of dementia after TBI was established using Cox proportional hazard models. There were about 43 thousands individuals with dementia, 10,164 individuals with a history of TBI, and 1.7 million controls. TBI was associated with increased risk of dementia including vascular dementia, unspecified dementia and AD[83].

Iacono D et al[84] concluded that a previous TBI exposure was a significant age-lowering factor for the onset of cognitive decline in either Alzheimer's Disease or non-Alzheimer's Disease conditions independent of sex, race, attained education or clinical diagnosis. Previous TBI exposures accelerated the onset of later cognitive decline across different brain diseases, with or without dementia. They analyzed data from the National Alzheimer's Coordinating Center [NACC] which provide information on history of TBI and longitudinal data on cognitive and non-cognitive domains for each available subject. They examined a total of 609 NACC subjects with a documented history of previous TBI of any type. They compared subject with or without a history of previous TBI at the time of their first cognitive decline assessment. The mean age of TBI-positive subjects was lower than the mean age of TBI-negative subjects at the time of their first cognitive decline assessment. In addition, neuropsychiatric and neurobehavioral symptoms were much more frequent in TBI-positive vs TBI-negative subjects.

In the Sariaslan et al paper[39], they identified and studied 1.3 million Swedish children born between 1973 and 1985 [12 years] for 41 years, and excluded children who could not be linked to both of their biological parents, who had died, or had migrated before the age of 26 years old, or

lacked data on parental sociodemographic factors, and adult outcome measures. The final sample was 1.1 million children. The study TBI patient was defined as an individual who had sought treatment for at least one episode of concussion [mild TBI] or moderate-to-severe TBI before 25 years as a TBI patient. The following outcomes and sequelae were studied: disability pension, specialist diagnoses of psychiatric disorders and psychiatric inpatient hospitalization, pre-mature mortality [defined as death before the age of 41 years old], low education attainment [defined as not having achieved secondary school qualifications] and receiving means-tested welfare benefits. There were 104,290 individuals who sustained TBI before the age of 25 years old. 77% of these patients, an overwhelming majority, suffered from mild TBI. TBI was associated with elevated risk of impaired adult functioning across all outcome measures. TBI contributed an absolute risk of 10% for specialist diagnoses of psychiatric disorders, low educational attainment, and welfare recipiency, and approximately 6% for disability pension. TBI patients were nearly 60% more likely to be hospitalized for any psychiatric disorder. Social functioning impairments were also 60% more likely in TBI patients than in unrelated controls; and 28% more likely to have attained a low level of education in adulthood, as well as receiving welfare benefits. Interestingly, these findings were not and could not be attributed to pre-existing or contemporaneous co-morbidities like neurological conditions and psychiatric disorders. The effects of TBI on the outcomes did not change materially over time. The age at first TBI, importantly, was a very strong moderator of poor functioning in adulthood with a positive association between older age at first TBI and all outcomes. Steffon Barber's previously attained intellectual and executive functioning has been significantly impaired. He can no longer become the underwater welder or anything similar that he had wanted to become. He is even expected to progressively deteriorate intellectually as time passes.

In the Fazel et al paper[38], they performed a 40-year study of 218,300 TBI patients and control-matched them with about 2.1 million members of the general population and 150,513 patient siblings without TBI. The patients had at least one patient episode of primary, secondary, or additional diagnoses. They excluded patients who died within 6 months of diagnoses of TBI to exclude immediate causes of death that could have caused the TBI. Compared to the general population, the unadjusted odds ratio of premature death among TBI patients was 3.6. Risks for all causes of premature death were elevated, with the largest causes from external factors including injuries, suicide, and assault. There were increased rates of psychiatric disorders in TBI patients including alcohol and drug use disorders and depression. The adjusted odds ratio for premature mortality in patients with both TBI and psychiatric comorbidity, specifically substance abuse and depression ranged from 8 to 24. Suicides in TBI patients like Steffon Barber had an odds ratio of 3.3 when compared to the general population.

In the Madsen et al paper[40] they studied nationwide registers in Denmark covering 7.4 million individuals from 1980 to 2014 and identified 567,823 TBI patients with a concussion [mild TBI], skull fracture or severe TBI [head injuries with evidence of structural brain injury]. Suicides recorded in the Danish Cause of Death register were identified. An overwhelming majority of the TBI patients, 423,502 were diagnosed with concussion [mild TBI]. Among the 34,529 suicides identified, 3536 [10.2%] had been diagnosed with TBI, including 2701 with concussion [mild TBI], 174 with skull fracture and 661 with severe TBI. The suicide rate for the general population was 19.9 per 100,000 person years; the suicide rate for TBI patients was 40.6 per 100,000 person years; the suicide rate for concussion [mild TBI] patients was 38.6 per 100,000 person years. The incidence rate ratio of suicide in TBI patients compared to the general population ranged from 2.64 to 3.35 for the basic research model. In their nation-wide cohort study, they confirmed that TBI patients have a significantly increased risk of suicides when compared to the general

population. Steffon Barber is more likely than not to exhibit remarkably increased risks of mood and psychiatric disorders including self-destructive behavior.

Steffon Barber stands an established risk of socio-economic deterioration and eventual homelessness following his TBI. This is an expected and known outcome of TBI[85-87]. The rates of TBI are significantly higher among individuals experiencing homelessness compared to the general population[86]. In a cross-sectional study design, purposive sampling was utilized to interview 115 English-speaking adults [ages 18-73] in two Colorado cities and 71% of total participants reported a significant history of TBI, and of these 74% reported a TBI prior to experiencing homelessness. Logistic regression models revealed a significant relationship between mental health and acquiring a TBI prior to experiencing homelessness[86].

In a systematic review and meta-analysis paper by Stubbs JL et al[85], they searched without date restrictions for original research studies in English that reported data on the prevalence or incidence of TBI, or the association between TBI and one or more health-related or function-related outcome measures. Studies were included whether they had a group or clearly identifiable subgroup of individuals who were homeless, marginally housed, or seeking services for homeless people. Of the 463 potentially eligible studies identified by the search, 38 studies were included in the systematic review and 26 studies were included in the meta-analysis. The lifetime prevalence of any severity of TBI in homeless and marginally housed individuals was 53.4% [95% CI]. TBI was consistently associated with poorer self-reported physical and mental health, higher suicidality and suicide risk, memory concerns, and increased health service use and criminal justice system involvement[85].

Hwang S et al[87] determined the lifetime prevalence of TBI and its association with current health conditions in a representative sample of homeless people in Toronto, Ontario. 601 men and 303 women at homeless shelters and meal programs were surveyed. TBI was defined as any self-reported head injury that left the person dazed, confused, disoriented or unconscious. The lifetime prevalence of TBI among homeless participants was 53% for any traumatic brain injury. For 70% of the respondents, their first TBI occurred before the onset of homelessness[87].

Having applied and analyzed these general causation principles which the case specific causation principles are based upon, Steffon Barber manifested novel, aggravating and progressive symptoms of brain damage, traumatic encephalopathy and dementia after his TBI. It is common knowledge that traumatic encephalopathy of all types can manifest with symptoms immediately after the occurrence of TBI, or after several hours, several days, several months, several years or several decades after the TBI. Steffon Barber's post-traumatic brain damage and dementia are permanent and are expected to be progressive as time goes by. His symptoms are permanent because TBI is a permanent injury given that the human brain is a post-mitotic organ and every injury to the brain is permanent. It is likely that Steffon Barber will remain wheelchair bound for the rest of his life. He is expected to develop the sequelae of significantly limited mobility like decubitus ulcers, urosepsis and sepsis.

The principles of the central limit theorem [CLT], which guide every measurable human index in science, mathematics, and statistics, dictate that there is a normal broad variation in the manifestations and symptomatic presentations of every disease including TBI. Every patient does not present exactly the same way. About 68%, 95% and 99.7% of all patients would present with indices that are within +/- 1, 2 and 3 standard deviations of the expectation, respectively. Therefore, if an interested party may claim that the manifestations and symptomatic

presentations of Steffon Barber may not be classical manifestations or presentations, this claim may not be scientifically valid since his manifestations and presentations would normally fall within +/- 1, 2 or 3 standard deviations from the expectation.
Steffon Barber has suffered from, and continues to suffer from multi-domain mood, cognitive, intellectual, behavioral, social, motor and somatic impairments pathognomonic of traumatic encephalopathy and dementia. Again, according to the CLT, each aspect of the multi-domain sequelae of TBI, as has been stated above, has been causally associated with exposure to TBI with a continuum and spectrum of causation risk. For example, while TBI has been confirmed to be a causal risk factor for developing various dementia and neurodegenerative diseases as has been stated above, Parkinson's Disease has exhibited one of the strongest and most compelling link to TBI as a causal risk factor amongst other spectrum of dementias and neurodegenerative diseases[25,49,52-57].

The two variables in this instance are TBI, the instigating, causal or risk factor, and persistent post-traumatic syndromes, traumatic encephalopathy and dementia, the outcome. The causal link between these two variables can be established based on the nine Hill's criteria for determining case specific causation[1,4]. The strength of the causal association between TBI and traumatic encephalopathy is very strong, which has been stated above. The epidemiological data linking these two variables are very consistent. The traumatic encephalopathy is temporally related to the TBI, in that the symptoms manifested after the TBI had occurred or where aggravated by the TBI. The biological gradient of TBI and TBI outcomes is well established in that the greater the exposure, the greater the likelihood of the sequelae[37,48,50,88-91]. The link between Steffon Barber's TBI and traumatic encephalopathy is scientifically plausible, reasonable and probable, given that the pathophysiological mechanisms of developing traumatic encephalopathy and dementia following TBI is well known. The cause-and-effect interpretation of TBI and traumatic encephalopathy and dementia does not seriously conflict and is coherent with the generally known facts of the history and biology of traumatic encephalopathy and dementia. There are both epidemiological and experimental evidence as has been presented above that TBI can be a causal, contributory, aggravating and accelerating risk factor for traumatic encephalopathy and dementia. Finally, there are other analogous cause and effect links between TBI and other diseases outside traumatic encephalopathy and dementia. Therefore, the case specific causation in the case of Steffon Barber is consistent with the general causation, and it can be concluded that the severe TBI he sustained from a gunshot wound of the head on April 27, 2021, was a single, independent, substantial, and significant contributory, aggravating and accelerating risk factor for his development of impaired motor functioning and hemiplegia, traumatic encephalopathy and dementia. It does not have to be the only mutually exclusive factor, but a significant and substantial risk factor by itself.

In the case specific causation analysis of Steffon Barber, we may consider the concept of co-morbidity in the medical sciences. For every disease, there are extenuating and aggravating factors, which can either decrease or increase the risk of suffering from or dying from a disease. A contemporaneous or co-morbid disease or factor that increases the risk of a second disease or factor does not denote causation, rather it denotes co-morbidity. Disease or event "A" that is co-morbid with disease or event "B" does not mean disease "A" causes disease "B" and vice versa.

Therefore, any assumed or presumed pre-morbid or contemporaneous occurrence of any other possible disease Steffon Barber may be suspected or alleged to have should be considered as a co-morbidity and not the cause of persistent and progressive symptoms of TBI, traumatic encephalopathy or dementia. This co-morbidity may have increased or decreased the risk of his

traumatic encephalopathy and dementia or may have synergistically and cumulatively aggravated his traumatic encephalopathy and dementia, but it did not cause or initiate his traumatic encephalopathy and dementia.
In conclusion therefore, yes, Steffon Barber suffered severe TBI on April 27, 2021 when he was shot in the head by a police officer. The TBI he sustained from this event at the age of 35-years-old is an independent, significant and substantial contributory, aggravating and accelerating factor for his neurodegenerative disease, dementia and traumatic encephalopathy.

Steffon Barber is already suffering from the signs and symptoms of a neurodegenerative disease following TBI- traumatic encephalopathy and dementia. His disease is permanent and progressive and is expected to progress to advanced dementia across decades. He is at a significantly and substantially increased risk of suffering from advanced young onset dementia [YOD] before the age of 65 years old within 20-30 years from the date of sustenance of severe TBI. He was 35 years old when he suffered severe TBI, therefore he is epidemiologically expected to develop severe and advanced dementia beginning at about 55 years old until about 65 years old. As he gets closer to 55 years old he is expected to need remarkably increasing levels of assistance with activities of daily living, medical attendance and specialized medical care. Beginning after the age of 55 years old up until about 65 years old, he is expected to become fully dependent on specialized assistance with activities of daily living and would need and become dependent on daily and full-time [24/7] basis on specialized medical attendance and medical care until the time of his death[78,79,81,92]. He is expected to remain wheelchair bound with increasingly progressive limitations and impairments of mobility for the rest of his life since the brain damage he has suffered is permanent and progressive as he gets older. He is also expected to suffer and manifest the commonly known sequelae of hemiplegia and impaired mobility like decubitus ulcers, urosepsis and sepsis.

3. Did Steffon Barber experience pain and suffering as a result of his severe TBI and gunshot wound of the head, and for how long?

It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of traumas and injuries and outcomes of traumas and injuries. The patterns of injuries generated by gunshots and all types of ballistics and the mechanisms of sustenance of these patterns of injuries are very well-established in the medical literature and are common knowledge. A specified prevailing pattern of trauma can reasonably predict the mechanisms of sustenance of the prevailing trauma.

Steffon Barber suffered severe TBI as a result of a gunshot wound of the head when he was shot by a police officer. Based on the prevailing forensic scenario, and on the generally accepted principles and common knowledge of medicine and science, and based on the global constellation, configurations, and anatomic conformations of the traumas sustained by Steffon Barber, he experienced pain and suffering beginning from the time of sustenance of his severe TBI and will continue to experience pain and suffering until his death. All forms of TBI, especially severe TBI, the type of TBI suffered by Steffon Barber, are permanent and progressive injuries.

Pathophysiology of conscious pain and suffering

Conscious pain and suffering are initiated by widespread free nerve endings situated in the skin, soft tissues, and organs. Pain can be elicited by multiple types of stimuli classified into three broad categories: mechanical, thermal, and chemical pain stimuli. Nerve endings for pain sensations generate electrical action potentials following all forms of tissue damage caused by all types of energies including, but not limited to, kinetic and mechanical energy from gunshots.

Action potentials are the sub-cellular physiologic basis for noxious conscious sensations and originate from voltage gated sodium and potassium electrolyte membrane pumps in the cell membranes of nerve cells, fibers, and synapses.

It takes few 10, 000$^{th's}$ of a second to generate action potentials. Action potentials are transmitted through nerve fibers to the brain. They are transmitted in peripheral nerves in the Aδ and C fibers for fast and slow pain respectively at impulse rates of 5-30 meters per second and 0.5-2 meters per second, respectively. There is therefore a double pain sensation, a fast-sharp pain, and a slow pain. The sharp pain apprises the person rapidly of imminent danger and prompts the person to react immediately and remove himself from the painful stimulus or imminent danger. The slow pain becomes greater as time passes resulting in continued intolerable pain and suffering prompting the person to continue to try to relieve the cause of the pain and flee from the imminent danger.

As an average human adult, experienced all types of gunshot and ballistics induced pain within milliseconds of contact and penetration of the bullet. One millisecond is one second divided into 1000 parts. For the slowest nervous mechanisms of pain sensation and consciousness, an adult male, like Steffon Barber, felt pain within 100 milliseconds.

Nerve pathways transmitting pain, terminate in the spinal cord. Secondary pathways transmit the pain from the spinal cord to the brainstem and thalamus, especially to the reticular activating system of the brainstem. From the thalamus, tertiary pathways transmit pain to other basal ganglia, limbic cortex, and neocortex of the brain. Pain stimuli are transmitted to the reticular nuclei of the midbrain, pons, and medulla; to the tectal midbrain and the periaqueductal gray matter. These lower regions of the brain, i.e., brainstem, are vital for the appreciation of the suffering types of pain.

Animals with their brains sectioned above the midbrain, to block any impulse reaching the neocortex and cerebral hemispheres, still experience suffering from pain caused by all types of trauma. Complete removal or disconnection of the somatosensory regions of the cerebral hemispheres, like we may have in quadriplegic patients, does not preclude a human or animal's ability to perceive and experience pain.

Pain impulses entering the spinal cord, brainstem and lower centers of the human brain can cause perception of pain. Pain perception is principally a function of the lower centers of the brain; however, the upper centers and cerebral hemispheres are responsible for the interpretation of the quality of pain and other cognitive aspects of pain, which are not needed to experience pain. Perception of pain is a primitive vegetative reflex similar to thirst and hunger.

The spinal reflex is the foundational basis for pain and suffering. As long as the spinal cord is intact, the human being will experience pain and suffering. This is buttressed by the fact that patients with high cervical spinal cord injuries and transections, and quadriplegia still experience pain and suffering in the body distal to the level of the spinal cord injury. Even with

the traumatic absence of any connectivity with the upper central nervous system, the cerebral hemispheres and brainstem, a patient will continue to experience pain and suffering driven by the spinal reflex. This is in part why patients who are quadriplegic experience pain and suffering in their bodies below the levels of the spinal cord injury based upon a variety of established pathophysiological mechanisms[93-103]. Therefore, in the absence of catastrophic spinal cord and cranial injuries, a traumatized patient would experience pain and suffering.

One of the factors we consider in the determination of general and case-specific causation is analogy. Is there another disease or trauma entity that is analogous to the case in question? And in this case an analogy is that of spinal cord injuries and quadriplegia. The quadriplegic patient can still experience pain and suffering below the level of the injury, which may include but are not limited to[97]:

1. Nociceptive pain
    a. Musculoskeletal pain
    b. Visceral pain
    c. Other nociceptive pain
2. Neuropathic pain
    a. At-level spinal cord injury pain
    b. Below-level spinal cord injury pain
3. Other pain
4. Unknown pain

Gunshot wounds elicit both the fast and slow pain types. Fast pain is felt within milliseconds while slow pain is felt within about one second. Following mechanical tissue damages, biochemical tissue reactants like bradykinin, serotonin, histamine, prostaglandins, leukotrienes, potassium ions, substance P, acetylcholine, acids, and proteolytic enzymes are expressed to elicit sustained secondary biochemical pain in addition to the primary fast pain directly caused by tissue damages. The biochemical pain elicited by these biochemical reactants is a slow type of suffering pain. The intensity of pain is closely correlated with the rate of tissue damage.

The brain is responsible for and sustains consciousness in human beings. The sensation of pain induces conscious suffering since pain is a noxious sensation, which stimulates the neocortex, limbic cortex, and forebrain to cause mental pain and suffering. All these neural processes occur in 1000th's of a second [milliseconds]. The human nervous system is one of the most efficient, effective, and optimal operating systems ever known to mankind. After centuries of empirical research mankind has not been able to fully decipher and reproduce the operating systems of the human brain and nervous system.

The human brain is a post-mitotic organ and can only survive on oxygen and glucose, which are supplied by blood that come from the heart, primarily in the internal carotid arteries and the vertebral arteries. While the brain is only about 2-3% of the body weight, it receives approximately 15% of the cardiac output at a rate of 750-900 ml/min of blood. The normal range of perfusion of the brain is about 50 to 65 ml/100 g/min [80-100 ml/100g/min for the gray matter and 20—25 ml/100g/min for the white matter, at a rate of oxygen consumption of 3.5 ml/100 g/min. The normal brain tissue partial pressure of oxygen is 35 to 40 mmHg. Brain tissue oxygen levels below 30 mmHg may cause brain tissue injury, and at 20 mmHg, the risk of brain damage becomes exponentially elevated. The threshold for brain infarction is 10-12 ml/100g/min of blood supply with neuronal injury and death beginning in 60 to 180 seconds.

Being a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself. This means that when the human brain suffers any type of irreversible injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy. There are so many types of brain injuries. For the human brain to suffer irreversible global brain injury and damage, there has to be an impaired supply of oxygen and blood to the brain. The established and generally accepted median or mean reference threshold time for irreversible hypoxic-ischemic brain damage to occur is 3 to 5 minutes in cumulative time. This means that irreversible brain damage can occur in less than 3 minutes or in more than 5 minutes, but with a mean or median time of close to 3 to 5 minutes.

Pain is a basic, vegetative and primitive human reflex with a primary objective of alerting the person to remove himself from imminent danger. Given that pain is a primitive reflex, patients who are alive but are suffering a disorder of consciousness still experience pain and suffering. There is no rigid demarcation between consciousness and unconsciousness. It is a continuum or spectrum of physiological functioning, however, there are broad varying degrees of disorders of consciousness with broad varying degrees of pain and suffering physiology and biochemistry[104-107]. We cannot reasonably differentiate or quantitate the degree of pain and suffering; rather it is a qualitative question of whether a person experiences pain or not. Therefore, pain and suffering are present in all persons with disorders of consciousness and should be adequately treated[105,108-111]. In the non-communicative, unconscious patient, the most relevant aspects of response to pain are physiologic (i.e., modification in the vital parameters such as heart rate and respiration) and behavioral (i.e., modification in the facial expression, motor and visual response)[112-114].

Steffon Barber's conscious pain and suffering

Steffon Barber's conscious pain and suffering sustenance began on April 27, 2021 when the bullet perforated his scalp. At this time, Steffon Barber was conscious and aware of his surroundings. His reticular activating center was completely intact and functional. The various domains of his brain and spinal cord, and cerebral and spinal functioning were intact and perceived the noxious stimuli within 1000th's of a second. His limbic system instigated high levels of primitive adrenergic fright-flight-fight response, which caused high levels of mental, somatic and biochemical pain and suffering.

He experienced mental, somatic, and biochemical pain and suffering from every tissue destruction he suffered as a result of his ballistics injuries. He suffered severe bodily injury and severe TBI. Transfer of forensically significant kinetic energy constituted noxious stimuli which generated novel action potentials, which traveled to the spinal cord and brain to cause novel mental, somatic, and biochemical pain, and suffering.

The biochemical secondary responses of this body to the primary tissue injuries and damages precipitated biochemical, anatomic, and pathophysiological noxious stimuli, which generated action potentials within 10,000ths of a second, which were transmitted to the spinal cord and to the brain to precipitate cumulative mental, somatic and biochemical pain, and suffering. The multimodal nature of the noxious stimuli resulted in synergistic and cumulative conscious experience of very high levels of mental, somatic and biochemical pain and suffering.

Action potentials from physical or thermal noxious stimuli eventually reach the limbic system to generate mental and psychological aspects of somatic pain and suffering. The primary injuries

initiated secondary tissue injuries, systemic and tissue reactive responses, which elicited novel biochemical pain and accentuated the conscious mental and somatic pain and suffering.

Following sustenance of his trauma and serious bodily injury, he began to develop bio-physiologic deficits and sequelae of his injuries. More nerve endings in his body, soft tissues and viscerae were recruited, many more action potentials were elicited and caused increasingly higher levels of mental, somatic, and biochemical pain and suffering.

Many more types of ions, peptides, proteins, and enzymes were expressed and activated, which enhanced his biochemical pain and suffering, which synergized with his pre-existing pain and suffering to cause higher, progressive mental, somatic, and biochemical pain, and suffering.

The brain is responsible for and sustains consciousness in human beings. The region of the brain responsible for consciousness is the brainstem. The center in the brainstem, which is responsible for consciousness, is the reticular activating system, which is deeply located in the central regions of the brainstem. As long as the reticular activating system remains anatomically and electrochemically intact, an individual like Steffon Barber will remain conscious and will experience pain and suffering. Following his sustenance of severe TBI Steffon Barber's reticular activating system did not suffer any catastrophic trauma, therefore it remained intact.

As time progressed, Steffon Barber continued to experience increasingly higher levels of mental, somatic, and chemical pain and suffering due to his secondary tissue injury cascades induced by the primary traumatic injuries. His pain and suffering persisted as he received emergency medical care and was transferred to the hospital. At the hospital he received both medical and surgical treatments and was eventually discharged. Medical and surgical intervention initiated novel cascades of mental, somatic and chemical pain and suffering, which contributed to the pre-existing mental, somatic and chemical pain and suffering. His severe TBI was permanent, persisted and metamorphosed into chronic TBI and traumatic encephalopathy.

Steffon Barber manifested and suffered from and continues to manifest and suffer from the established impairments, symptoms and signs of serious bodily injury, acute severe TBI, chronic TBI and traumatic encephalopathy. He continues to experience varying modalities of conscious mental, somatic and chemical pain and suffering from the permanent, persistent, progressive and chronic TBI, the long-term sequalae of the gunshot wound he suffered. He continues to experience varying modalities of conscious mental, somatic and chemical pain and suffering from the medical and surgical interventions and treatments he received and continues to receive for this permanent brain damage and chronic TBI. The somatic symptoms and signs, motor symptoms and signs, mood symptoms and signs, behavioral symptoms and signs, cognitive and executive functioning symptoms and signs of the sequelae of his gunshot wound of the head and TBI continue to generate neurological, patho-physiological and biochemical cascades for mental, somatic and chemical pain and suffering.

The sequelae of his TBI are permanent and shall continue for the rest of his life until death. Since he was shot on April 27, 2021, Steffon Barber has suffered and experienced conscious pain

and suffering from his TBI and permanent sequelae of his TBI for 4-5 years[3,4]. He will continue to suffer and experience conscious pain and suffering from his TBI and permanent sequelae of his TBI until death[5].

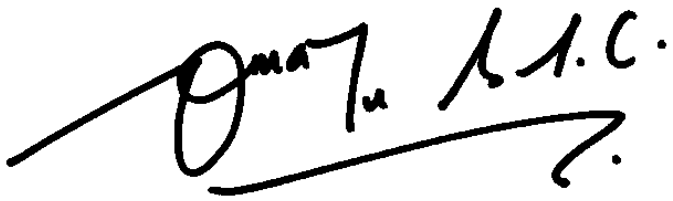
The human body continues to experience debilitating trauma-induced and physiologic biochemical pain and suffering until there is a complete cessation of all bodily functions and death as long as the spinal and cranial reflexes are intact.

I have provided my opinions and conclusions with a reasonable degree of medical and scientific certainty.

I reserve the right to amend, supplement, revise and/or modify my opinions and report, up to the time of trial, should additional information become available

Thank you.

Very truly yours,



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[3] Medicine is not an absolute science, and these estimated ranges should not be interpreted as absolute quantitative estimations of time. Quantitative ranges of any measurable index are common practice and are the standard of practice in pathology and medicine.

[4] Human events like loss of consciousness and death involve a continuum of pathophysiological events on the cellular and gross functional levels without any identifiable rigid transitions or demarcations. Therefore, the determination of the time of occurrence of these events are guided by the time the events have been reproducibly and quantifiably confirmed. For example, the time of death of any individual is determined by the time the individual was pronounced dead by a designated medical professional who has clinically assessed the patient and confirmed the patient to be dead based on prevailing, reproducible and quantifiable clinical evidence that the patient was dead.

[5] Medicine is not an absolute science, and these estimated ranges should not be interpreted as absolute quantitative estimations of time. Quantitative ranges of any measurable index are common practice and are the standard of practice in pathology and medicine.

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110. Pistoia F, Sacco S, Stewart J, Sara M, Carolei A. Disorders of Consciousness: Painless or Painful Conditions?-Evidence from Neuroimaging Studies. *Brain Sci*. Oct 8 2016;6(4)doi:10.3390/brainsci6040047

111. Fins JJ, Shapiro ZE. Pain Management, Disorders of Consciousness, and Tort Law: An Emergency Tort to Fix a Longstanding Injustice. *Indiana Law Journal*. 2023;98(3)(1):693–720.
112. Riganello F, Soddu A, Tonin P. Addressing Pain for a Proper Rehabilitation Process in Patients With Severe Disorders of Consciousness. *Front Pharmacol*. 2021;12:628980. doi:10.3389/fphar.2021.628980
113. Bodien YG, Allanson J, Cardone P, et al. Cognitive Motor Dissociation in Disorders of Consciousness. *N Engl J Med*. Aug 15 2024;391(7):598–608. doi:10.1056/NEJMoa2400645
114. Naro A, Bramanti P, Bramanti A, Calabro RS. Assessing pain in patients with chronic disorders of consciousness: Are we heading in the right direction? *Conscious Cogn*. Oct 2017;55:148–155. doi:10.1016/j.concog.2017.08.009

CURRICULUM VITAE AND BIBLIOGRAPHY

PART 1: CURRICULUM VITAE

BENNET I. OMALU, MD, MBA, MPH, CPE, DABP-AP,CP,FP,NP

Anatomic Pathologist/ Clinical Pathologist/ Forensic Pathologist/ Neuropathologist/ Epidemiologist
Clinical Professor of Medical Pathology and Laboratory Medicine, University of California, Davis [UC-Davis]
President and Medical Director, Bennet Omalu Pathology [BOP], Inc.

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Sacramento, CA 95864

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Honorary Doctorate Degrees:

1. Doctor of Science
   Westminster College
   Fulton, Missouri, September 15, 2016

2. Doctor of Science
   Xavier University of Louisiana
   New Orleans, Louisiana, May 7, 2016

3. Doctor of Science
   Royal College of Surgeons in Ireland
   Dublin, Ireland, June 7, 2017

Degrees and Certifications:

1. Board Certification in Anatomic Pathology (AP)
   Diplomate, American Board of Pathology
   American Board of Pathology, July 2002.

2. Board Certification in Clinical Pathology (CP)
   Diplomate, American Board of Pathology
   American Board of Pathology, July 2003.

3. Board Certification in Forensic Pathology (FP)
   Diplomate, American Board of Pathology
   American Board of Pathology, November 2004.

4. Board Certification in Neuropathology (NP)
   Diplomate, American Board of Pathology
   American Board of Pathology, September 2005.

5. Board Certification in Medical Management
   Certified Physician Executive [CPE]
   Certifying Commission in Medical Management
   American College of Physician Executives, February 2011

6. Masters in Public Health (MPH), Epidemiology
   University of Pittsburgh, Graduate School of Public Health
   Pittsburgh, Pennsylvania, April 2004.

7. Masters in Business Administration [MBA]
   Tepper School of Business, Carnegie-Mellon University
   Pittsburgh, Pennsylvania, May 2008.

8. One year clinical Internship Certificate, general/ family practice
   University of Nigeria Teaching Hospital, Enugu, Nigeria, January 1992.

9. Bachelor of Medicine and Bachelor of Surgery (M. B., B. S.)
University of Nigeria, Enugu, Nigeria, June 1990.

10. United States Medical Licensing Examinations (Steps I and II), September 1993.

11. United States Medical Licensing Examination Step III, May 1998.

12. Grade 1 certificate in the Theory of Music, The Royal Schools of Music, London, England, 1981.

Professional and Post-Graduate Education and Training

1.	College of Medicine, University of Nigeria, Enugu, Nigeria.	July 1984 - July 1990
2.	House Physician, Department of Pediatrics, Enugu General Hospital, Enugu, Nigeria.	August 1990 - Dec. 1990
3.	Internship in General/ Family Practice, University of Nigeria, Hospital, Enugu, Nigeria.	January 1991 - January 1992
4.	Emergency Room Physician, Jos University Hospital, Jos, Nigeria.	March 1992 - October 1994
5.	Visiting/ Research Scholar, Cancer Epidemiology, Dept. of Epidemiology, School of Public Health, University of Washington, Seattle.	October 1994 - June 1995
6.	Pathology Residency, Anatomic/ Clinical College of Physicians and Surgeons of Columbia University, Harlem Hospital Center, New York City.	July 1995 - June 1999
7.	Fellowship training in Forensic Pathology, Allegheny County Coroner's Office University of Pittsburgh, Pittsburgh, Pennsylvania.	July 1999 - June 2000
8.	Fellowship training in Neuropathology, University of Pittsburgh Medical Center University of Pittsburgh, Pittsburgh, Pennsylvania.	July 2000 - June 2002
9.	Masters in Public Health: Epidemiology Graduate School of Public Health University of Pittsburgh, Pittsburgh, Pennsylvania	June 2002 – April 2004

10. Masters in Business Administration
Tepper School of Business
Carnegie Mellon University, Pittsburgh, Pennsylvania August 2005 – May 2008

Medical Licensure

1. License of practice as a physician, unrestricted, State of Indiana 1998 - 2023
2. License of practice as a physician, unrestricted, State of Pennsylvania 1998 - 2023
3. License of practice as a physician, unrestricted, State of Hawaii 2003 - 2023
4. License of practice as a physician, unrestricted, State of California 2007 – Present

Professional Medical Affiliations and Memberships

1. Member, American Association of Physician Leadership, 2006 – present
2. Member, American Association for the Advancement of Science [AAAS], 2005 – present
3. Fellow, College of American Pathologists (CAP), 1995 – present
4. Fellow, American Society of Clinical Pathologists (ASCP), 1995 – present
5. Member, American Association of Neuropathologists (AANP), 2002 – present
6. Member, National Association of Medical Examiners (NAME), 1999 – present
7. Member, American Academy of Forensic Sciences, 2004 – present
8. Member, United States and Canadian Academy of Pathology, 2003 – present
9. Member, International Academy of Pathology, 2003 – present
10. Fellow, American College of Epidemiology, 2004 – present
11. Member, American Association of Public Health Physicians, 2004 – present
12. Member, American Public Health Association, 2004 - present
13. Member, American Society for Investigative Pathology, 2004 - present
14. Member, American Medical Association (AMA), 2002 - present
15. Member, San Joaquin Medical Society, 2009 – present
16. Member, California Medical Society, 2009 – present
17. Member, California Society of Pathologists, 2015 - present
18. Member, Union of American Physicians and Dentists, 2007 - present
19. Member, Pennsylvania Medical Society, 2003 - 2007

20. Member, Allegheny County Medical Society, 2003 – 2007
21. Member, New York County Medical Society, 1997 - 1999
22. Member, Nigerian Medical and Dental Council, 1990
23. Member, Association of Nigerian Physicians in the Americas, 2017 - present

Appointments

1. Consulting Forensic Pathologist-Neuropathologist, Kaiser Permanente Hospitals, Central California, February 2018 – Present.
2. Physician Associate and Professor, Department of Medical Pathology and Laboratory Medicine, University of California, Davis, February 2018 – Present.
3. Consulting Forensic Pathologist-Neuropathologist, Yosemite Pathology Medical Group, January 2018 – Present.
4. Consulting Forensic Pathologist-Neuropathologist, Lake County, California, February 2018 - Present.
5. Visiting Professor, Emory University, Department of Pathology and Laboratory Medicine, March 2017.
6. Clinical Professor, Department of Medical Pathology and Laboratory Medicine, University of California, Davis, January 2017 – Present.
7. Co-Director/Co-Founder, Taumark, Inc, Better Brain Diagnostics, May 2013 – present.
8. Chief Medical Examiner, Washington, D.C. Appointed on October 11, 2013. Declined Offer.
9. Co-Director, Brain Injury Research Institute, West Virginia University/NorthShore University Health System, November 2009 – present.
10. Visiting Professor, Department of Neurosurgery, West Virginia University, Dec. 2009 – Dec. 2011
11. Member, NFL Players Association Concussion and Traumatic Brain Injury Committee, December 2009 – 2012.
12. Visiting Professor, Rockefeller Institute of Neuroscience, Morgantown, Virginia, August 2009 – April 2010.
13. Chief Medical Examiner, San Joaquin County, California, September 2007 – March 2018.
14. Associate Clinical Professor, Department of Medical Pathology and Laboratory Medicine, University of California, Davis, September 2013 – January 2017.

15. Clinical Professor of Pathology, Department of Pathology, University of California, Davis, March 2012 – July 2013.
16. Associate Physician Diplomate, Medical Pathology and Laboratory Medicine, University of California-Davis Medical Center, March 2012 – July 2013.
17. Associate Clinical Professor of Pathology, Department of Pathology, University of California, Davis, May 2008 – January 2012.
18. Adjunct Assistant Professor of Pathology, University of Pittsburgh, Pittsburgh, Pennsylvania, June 2010 – 2012.
19. Assistant Clinical Professor of Epidemiology, Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, March 2004 – 2008
20. Clinical Associate Professor of Pathology and Clinical Instructor, Department of Pathology, University of Pittsburgh, Pittsburgh, Pennsylvania, May 2003 – 2009
21. Attending Forensic Pathologist, Allegheny County Coroner's Office, July 2002 – March 2007
22. Attending Forensic Neuropathologist, Allegheny County Coroner's Office, July 2002 – March 2007
23. Associate Forensic Pathologist, Allegheny County Coroner's Office, July 2000 – June 2002

Hospital Admissions and Practice Privileges

1.	University of California, Davis, Health System/ Medical Center Department of Medical Pathology and Laboratory Medicine 2315 Stockton Blvd Sacramento, CA 95817	March 2012 to July 2013 February 2018 to Present
2.	Contra Costa Regional Medical Center Department of Pathology 2500 Alhambra Avenue Martinez, CA 94553	January 2013 to Present

3. San Joaquin General Hospital — September 2007 to Present
   Department of Pathology
   500 West Hospital Road
   French Camp, CA 95231

4. Yosemite Pathology Medical Group — August 2017 to Present
   Business Associate- Autopsy Services
   2625 Coffee Road
   Modesto, CA 95355

5. NorthBay Medical Center — June 2018 to Present
   1200 B. Gale Wilson Boulevard
   Fairfield, CA 94533

6. Adventist Health Bakersfield Hospital — 2022 to Present
   2615 Chester Avenue
   Bakersfield, CA 93301

Journal Editorial and Peer Review Experience

1. Associate Editor, Neurosurgery Journal, Sports and Rehabilitation Section, September 2009 – 2014
2. Reviewer, Militarily Relevant Peer Reviewed Alzheimer's Disease Research Program (MRPRA), 2012: American Institute of Biological Sciences [AIBS], Scientific Peer Advisory and Review Services Panel.
3. Reviewer, Nigerian Journal of Clinical Practice, February, 2011 to 2014
4. Reviewer, Nigerian Journal of Surgery, October 2011
5. Reviewer, The Journal of Neuropsychiatry and Clinical Neurosciences, April 2012
6. Reviewer, The Journal of Forensic Nursing, June 2009, October 2012, February 2013
7. Reviewer, Forensic Science, Medicine and Pathology, November 2012

Consulting and Peer Review Assignments

1. Consulting Forensic Pathologist/ Neuropathologist, Cyril H. Wecht and Pathology Associates Inc. June 2000 – January 2006.
2. Consulting Forensic Neuropathologist, Office of the Chief Medical Examiner, Commonwealth of Virginia: August 2006 – December 2012
3. Consulting Forensic Neuropathologist, Office of the Coroner/Medical Examiner, Clark County, Las Vegas, Nevada, September 2007 – December 2012
4. Consulting Forensic Neuropathologist, Office of the Coroner/Medical Examiner, Washoe County, Reno, Nevada, 2009 – present
5. Consulting Forensic Pathologist, Solano County Coroner's Office, California, 2011 - 2012
6. Consulting Neuropathologist, Conemaugh Memorial Hospital, Johnstown, Pennsylvania: August 2003 – August 2007
7. Consulting Expert Witness/Forensic Pathologist/Neuropathologist, Public Defender's Office, County of Sacramento, California, April 2010 – present.
8. Consulting Forensic Neuropathologist, Medical Examiner's Office, Allegheny County, Pittsburgh, Pennsylvania, January, 2011.
9. Consulting Forensic Neuropathologist, Medical Examiner's Office, County of Summit, Akron, Ohio. October, 2011.
10. Consulting Forensic Neuropathologist, American Forensics, Dallas, Texas. October, 2011.

Congressional Hearing, Testimony and Briefing

The Congress of the United States

1. One Hundred and Eleventh Congress, Congress of the United States, House of Representatives, Committee on the Judiciary, Field Hearing: Legal Issues Relating to Football Head Injuries, Part II, Monday, January 4th, 2010.

2. One Hundred and Eleventh Congress, Congress of the United States, House of Representatives, Committee on the Judiciary, Judiciary Forum: Head and Other Injuries in Youth, High School, College and Professional Football, Monday, February 1, 2010.

3. One Hundred and Fourteenth Congress, Congress of the United States, House of Representatives: Dr. Omalu's Briefing on his Work and Legacy. Energy and Commerce Committee Room, 2123 Rayburn HOB. Tuesday, January 12, 2016.

California State Assembly

1. Testimony in support of AB 2007- Concussion Management for Youth Sports Leagues, Committee on Arts, Entertainment, Sports, Tourism and Internet Media, Tuesday, May 3, 2016.

2. Testimony in support of SB 1303- An act to amend Sections 24000, 24009, and 24010 of the Government Code, relating to local government [Coroner: County Office of the Medical Examiner], Senate Committee on Governance and Finance, California State Senate, April 11, 2018.

3. Testimony in support of SB 1303, Senate Committee on Public Safety, California State Senate, April 24, 2018.

4. Testimony in support of SB 1303, Assembly Committee on Local Government, California State Assembly, June 20, 2018.

5. Testimony in support of SB 1303, Assembly Committee on Public Safety, California State Assembly, June 26, 2018.

New York State Assembly

1. Testimony in support of A4448- An act to amend the public health law, in relation to prohibiting children thirteen years old and younger from playing tackle football. Roundtable discussion before Concussion movie screening to New York State Assembly and New York State Senate Members. Wednesday, May 4, 2016.

Selected Honors, Awards, Recognition and Achievements

1. 2019 Medgar Evers Freedom Fighter Award, National Association for the Advancement of Colored People [NAACP], Stockton, California, November 16, 2019, Stockton, California.

2. Distinguished Alumnus- Medical Discovery Award, College of Medicine, University of Nigeria, Enugu, Nigeria, September 19, 2019.

3. Recognized as a member of the Trailblazer of the Lab Power List, 2019. The Pathologist, August 2019. https://thepathologist.com/power-list/2019

4. Recognized as one of the "100 Figures Who Shaped The NFL's First Century": "Perhaps the single most significant name in football post-2000, Bennet Omalu—the Nigerian-born doctor who first discovered evidence of brain trauma in the body of former Steelers great Mike Webster—changed the way everyone from players and coaches to administrators and concerned parents have approached the violent sport". 100 Figures Who Shaped the NFL's First Century. By The Sports Illustrated Staff, August 28, 2019. https://www.si.com/nfl/2019/nfl-100-most-influential-figures-all-time

5. 2019 Peace And Justice Award, California State University, Sacramento, College of Health and Human Services, Center for African Peace and Conflict Resolution, Sacramento, April 27, 2019.

6. Leadership Award, Carson Scholars Fund, April 13, 2109, San Diego, California.

7. Pioneer Award, 2019 Champions of Health Awards, National Medical Fellowships, Bay Area, March 2, 2019, Berkeley, California.

8. Appointed Member, Traumatic Brain Injury Advisory Board, California Department of Rehabilitation, State of California Health and Human Services Agency, January 1, 2019 to May 31, 2021.

9. Department of the Army, California Army National Guard, Sacramento, California, Nigerian 68W Familiarization/SES Donald Get [AFRICOM] Visit to California, Invitational Appearance Based on Military Necessity, August 27, 2018.

10. William Steiger Memorial Award, 2018: American Conference of Governmental Industrial Hygienists [ACGIH]: For Efforts Contributing to the Advancement of Occupational Health and Safety. March 2018.

11. Honorary Member Award, Academy for Sports Dentistry, June 23, 2017, San Francisco, California.

12. Residents Choice Award, 2017, Emory University School of Medicine, Department of Pathology and Laboratory Medicine, March 13, 2017.

13. Joint Members Resolution No. 116 to Commend Dr. Bennet Omalu for Exemplary Record of Medical Achievements, State of California Legislature, February 22, 2017.

14. Certificate of Recognition, Board of Supervisors, San Joaquin County, for contributions and distinguished service to San Joaquin County, January 10, 2017, Stockton, California.

15. Outstanding Achievement in Medicine Award, The San Joaquin Medical Society, Stockton, California, December 15, 2016.

16. Rescuer of Humanity Award, 2016, Values-in-Action Foundation, Cleveland, Ohio, December 6, 2016.

17. Service To Science Award, 2016 D. Walter Cohen, DDS Award, NDRI [National Disease Research Interchange], November 18, 2016, Philadelphia, Pennsylvania.

18. Distinguished Service Award [The highest award of the American Medical Association-AMA], November 12, 2016, AMA Interim Meeting, 2016, Orlando, Florida.

19. Beacon of Ethics Award, The Greater Omaha Alliance For Business At Creighton University, Better Business Bureau Annual Integrity Awards Luncheon, November 2, 2016.

20. Healing Hand Award, The El Paso Hispanic Chamber of Commerce, 26th Anniversary Fiesta Celebration and Pinata Bash, October 15, 2016.

21. Honorary Lifetime Membership, The National Honor Society in Neuroscience [Nu Rho Psi], University of Evansville, Indiana, October 13, 2016.

22. Invited Guest and Speaker, Camp Fire 2016, Santa Barbara, California, September 29 – October 2, 2016.

23. Special Tribute to Bennet Omalu, State of Michigan, The Ninety-Eighth Legislature, At Lansing, September 20, 2016.

24. The Louise Blouin Foundation Award for a Positive Impact on a Global Scale, The Eleventh Annual Awards, The Blouin Creative Leadership Summit Awards Reception, September 19, 2016, New York City.

25. Above and Beyond Award, 2016. The Network of Ethnic Physician Organization and The California Medical Association Foundation, September 17, 2016.

26. The 2016 Green Lecture Speaker, Westminster College, Fulton, Missouri, September 15, 2016.

27. 2016 Distinguished Great Immigrant, Carnegie Corporation of New York, June 30, 2016.

28. 2016 Dr. Ernst Jokl Sports Medicine Award, Unites States Sports Academy, June 29, 2016.

29. Key Note Speaker, 2016 Commencement, University of California, Davis, School of Medicine, May 28, 2016.

30. Honorary Fellow, American Association of Physician Leadership, April 15, 2016, Washington, DC.

31. Invited Speaker, W.L. Mellon Speaker Series, April 14, 2016, Tepper School of Business, Carnegie Mellon University, Pittsburgh, Pennsylvania.

32. Pioneer in Medicine Award, 2016, The Society for Brain Mapping and Therapeutics (SBMT), April 9th, 2016, 13th Annual World Congress for Brain Mapping and Therapeutics, Miami, Florida.

33. 2016 Red Cross Heroes-Spirit of the Red Cross Award. The American Red Cross, Gold Country Region. March 16, 2016.

34. Contribution to Science and Education Award, 37th Annual Young Artist Awards, The Young Artist Foundation, Los Angeles, California, March 13, 2016.

35. Congressional Honors, United States Congress, House of Representatives, for the discovery of Chronic Traumatic Encephalopathy and Raising Awareness on Repetitive Brain Trauma, Wednesday, February 3, 2016, Congressman Jerry McNerny, D-California, 9th District.

36. Invited to the Address to the Congress on the State of the Union, President Barack Obama, January 12, 2015, 114th Congress, United States House of Representatives, Guest of Honorable Jackie Speier, U.S. Representative for California's 14th Congressional District.

37. Invited Guest, 73rd Golden Globe Awards, The Hollywood Foreign Press Association, January 10, 2016, Beverly Hills, California.

38. Presented the Key to the City of Lodi by Mark Chandler, Mayor, City of Lodi, California, January 9, 2016.

39. Invited Guest, 7th Annual Governors Award, Academy of Motion Pictures Arts and Sciences, November 14, 2015, Hollywood, California.

40. Medscape Best Physicians of the Year: 2015.

41. WebMD Health Heroes Award- Scientist, 2015. November 5, 2015.

42. Founding member and director, Bennet Omalu Foundation, Inc., September 2014.

43. Member, Greater Talent Network Speakers' Bureau, New York, New York, 2015

44. Recognized as one of the "120 Great Nigerians You Never Knew". First Bank of Nigeria [since 1894], Ed Emeka Keazor, MME Media, Johannesburg, South Africa, 2014.

45. Certificate of Special Congressional Recognition, San Joaquin County Bar Association, Law Day Luncheon. April 30, 2015. Jerry Mcnerney, Member of the United States Congress.

46. Named among 20 Top Forensic Pathology Professors Online. Forensics Colleges. November 2013. [http://www.forensicscolleges.com/blog/profs/20-top-forensic-pathology-professors; http://www.ucdmc.ucdavis.edu/publish/news/newsroom/8426]

47. 2013 Alumni Achievement Award, University of Nigeria Alumni and Friends Association, U.S.A. Saturday, October 19, 2013, Los Angeles, California.

48. Founding Member, CTEM, Inc. Wheeling, West Virginia, 2013.

49. Nominated for the Potamkin Prize for Research in Pick's, Alzheimer's, and Related Diseases, American Academy of Neurology, 2012.

50. Years of Excellence In Pioneering Neuropathology Concussion Research For Athlete Populations. June 22, 2012, Sports Concussion Institute, Sixth Annual National Summit On Sports Concussion And Other Athletic Injuries.

51. America's Top Physicians, 2007, 2008, 2009, 2010, 2011. Consumer's Research Council of America, Washington, DC.

52. Member, Board of Directors, Brain Injury Research Institute, West Virginia University, Morgantown, West Virginia, August 2010 to present.

53. Discovered and named Chronic Traumatic Encephalopathy [CTE] in American football players and American professional wrestlers, 2002 and 2007.

54. Co-Founder, Brain Injury Research Institute, West Virginia University, Morgantown, West Virginia, November 2009.

55. First Chief Medical Examiner of San Joaquin County, California.

56. Honored Visiting Professor, Department of Neurosurgery, West Virginia University, October 1, 2008.

57. Founder and President, Bennet Omalu Pathology, Inc. October, 2011.

58. Identified the West Nile Virus within the neurons of an infected deceased man using fluorescent-labeled con-focal immuno-microscopy, 2004.

59. The 2002 'Manifesting the Kingdom' Award, Most Reverend Donald W. Wuerl, Bishop, Catholic Diocese of Pittsburgh January 5, 2003.

60. National merit list, Masters in Public Health, Graduate School of Public Health, University of Pittsburgh, April 2004.

61. Honorary Fellowship Award, American Registry of Pathology at the Department of Environmental and Toxicologic Pathology, Armed Forces Institute of Pathology (AFIP). October, 1998.

62. College of American Pathologists (CAP) Foundation Informatics Award: Automated Information Management in the Clinical Laboratory. (Ann Arbor, Michigan) May 1998.

63. World Health Organization Research scholar Award, Cancer Epidemiology, School of Public Health, University of Washington, Seattle, Washington. October 1994 - June 1995.

64. Chief Resident, Department of Pathology, Harlem Hospital Center July 1998 - June 1999.

65. Harlem Hospital House Staff President, March 1998 - April 1999.

66. Laboratory Inspector, College of American Pathologist (CAP), Stanford University, November 2002.

67. New York state delegate, College of American Pathologists Residents Forum, July 1997 – June 1999.

68. Resident Member, Harlem Hospital Medical Board, April 1998 - March 1999.

69. Resident Member, Harlem Hospital Community Board, April 1998 - March 1999.

70. National Delegate, Committee of Interns and Residents (CIR), March 1996 - April 1999.

71. Harlem Hospital Residency Program Liaison, American Society of Clinical Pathologists, July 1997 - June 1999.

72. Honorable Mention, House Staff Research Fair, College of Physicians and Surgeons of Columbia University at Harlem Hospital Center, New York, for: An Immunohistochemical Profile of Tumor Associated Antigens in Malignant Melanoma and Benign Melanocytic Nevi: CD44, p53 protein, Cathepsin B, Melan-A Gene Product. April 1998.

73. Honorable Mention, House Staff Research Fair, College of Physicians and Surgeons of Columbia University at Harlem Hospital Center, New York, for: Forensic implications of the Analytical Evaluation of Human Tissues after Exposure to Cesium Chloride: Atomic Absorption Spectrometry. May 1999.

74. College Scholarship award: National Merit List, Federal Government of Nigeria. September 1979 - July 1984.

75. Producer, “Christ is King !! A Liturgical Selection”: an audio compact disc album for Saint Benedict the Moor Roman Catholic Church, Pittsburgh, Pennsylvania. November 2003.

76. President and Founder/ Principal partner, BOGE LLP, a health management and consulting company, Pittsburgh, Pennsylvania, March 2004 – 2007.

77. President and Founder/ Principal partner, Clairton Community Cultural Center, a community re-development company, Pittsburgh, Pennsylvania, 2005 – 2007.

78. President and Founder/ Principal partner, Clairton Community Health Center, a 24-hour community primary care health center, Pittsburgh, Pennsylvania, 2005 – 2007.

79. Trustee, Prostate Health and Research Foundation, a non-profit prostate health education organization, Lagos, Nigeria, March 2004 – 2007.

Selected Invited Lectureships and Talks

1. Guest Speaker, AD in 3D- An Immersive Tour of Alzheimer's Disease and Modifiable Risk Factors. Marriott Marquis San Diego Marina, San Diego, California. April 7, 2025, Eli Lilly.

2. Guest Lecturer, Mental Effects of TBI/ CTE, Grand Rounds, Hackensack University New Jersey, Hackensack Meridian Health, Mental effects of TBI / CTE, March 19, 2025.

3. Keynote Speaker, Black Health Matters Conference 2023: "Decolonizing Health: Recentering Black Wellness and Community Empowerment", February 19, 2023, Harvard Undergraduate Black Health Advocates, Harvard University, Cambridge, Massachusetts.

4. Business Ethics Class, Dean Hanumantha R. Unnava, UC Davis Graduate School of Management, Davis, California, February 10, 2020.

5. Guest Speaker, Board of Trustees Meeting, Educational Commission for Foreign Medical Graduates [ECFMG]/ Foundation for Advancement of International Medical Education and Research [FAIMER], December 5, 2019, Philadelphia, Pennsylvania.

6. Dreamforce Fireside Chat: Bennet Omalu and Emilia Clarke, November 22, 2019, San Francisco, California, Salesforce.

7. Keynote Speech: "I practice my faith in my science- by faith the impossible becomes possible". Ministry Days 2019 convention, the Catholic Diocese of Sacramento, September 28, 2019, Sacramento, California.

8. Keynote Speech: "Truth Doesn't Have a Side". 2019 Annual Member Symposium, Beta Healthcare Group, La Jolla, California, September 26, 2019.

9. Commencement Speech, UC Davis Graduate Studies, 72nd Annual Commencement, June 13, 2019, ARC Pavilion, University of California, Davis.

10. Keynote Speech, "Don't Break the Rules, Change the Game", Courage at Our Core initiative, Progressive Group of Insurance Companies, Mayfield Village, Ohio, June 12, 2019.

11. Keynote Speech, "An Afternoon with Dr. Bennet Omalu", 2019 OhioHealth Neuroscience Symposium, May 17, 2019, OhioHealth, Columbus, Ohio.

12. Keynote Speech, "A morning with Bennet Omalu", Values Day, May 10, 2019, Annual Conference, McKinsey & Company, Chicago, Illinois.

13. Distinguished Keynote Speaker, 28th Annual Africa Peace Awards, The Center for Africa Peace and Conflict Resolution, College of Health and Human Services, California State University, Sacramento, April 27, 2019.

14. Keynote Speaker, 2019 Annual Banquet, Carson Scholars Fund, San Diego, California, April 13, 2019.

15. Keynote Speaker, 2019 Cal-HOSA [Health Occupations Students of America] State Leadership Conference, Sacramento Convention Center, March 30, 2019.

16. California Military Department/ California National Guard, Key Note Speaker and Certificate of Appreciation, February 2019 Special Emphasis Observance, February 25, 2019, Sacramento, California.

17. "Don't Break the Rules, Change the Game: How Bennet Omalu Single-Handedly Changed American Football, Professional Sports, and How the World Perceives Traumatic Brain Injuries". American Academy of Neurology Annual Meeting, Los Angeles, California, April 25, 2018.

18. Keynote Speech: Conformational Intelligence and Innovation, 2018 MedHealth Summit, Michigan, United States, and Ontario, Canada. Detroit, Michigan, April 19, 2018.

19. "An Afternoon with Dr. Bennet Omalu". Florida Justice Association, World Center Marriot Hotel, Annual Meeting, Orlando, Florida, March 23, 2018.

20. "An Evening with Dr. Bennet Omalu". The Hinman Dental Society, Georgia World Congress Center, Atlanta, Georgia, March 22, 2018.

21. "NFLPA Denver Chapter Movie Night, "Concussion". Sports Legend Assistance Fund in affiliation with NFLPA Denver Chapter, Landmark Theater, Greenwood Village, Colorado, November 9, 2017.

22. "Don't Break the Rules; Change the Game". Tulsa Town Hall, Tulsa Performing Arts Center, Tulsa, Oklahoma, October 27, 2017.

23. "An Evening with Dr. Bennet Omalu". Baraboo Growth, LLC, Milwaukee Club, Milwaukee, Wisconsin, October 24, 2017.

24. "American Football, Player Safety and Health, and Future of Contact Sports". Chicago Ideas, Morningstar, Chicago, Illinois, October 21, 2017.

25. "An Evening with Dr. Bennet Omalu". Commonwealth Club Present Dr. Bennet Omalu, The Commonwealth Club, San Francisco, California, August 23, 2017.

26. "Don't Break the rules, Change the Game: How Bennet Omalu single-handedly changed American football, professional sports, and how the world perceives traumatic brain injuries". Keynote

Address, Life Chiropractic College West, Wave 2017 Conference, San Francisco, California, August 5, 2017.

27. Keynote/Commencement Speaker, Conferring Ceremony, School of Medicine, Royal College of Surgeons in Ireland, Dublin, Ireland, June 7, 2017.

28. "An Evening with Bennet Omalu". North Shore University Hospital Medical Staff Society, 2017 Dinner and Awards Ceremony. Garden City, New York, May 4, 2017.

29. "An Evening with Bennet Omalu". University of North Carolina- Wilmington, Leadership Lecture, Wilmington, North Carolina, March 29, 2017.

30. "Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American Football, Professional Sports, and how the world perceives traumatic brain injuries." Frederick Speaker Series, Weinberg Center for the Arts, Inc. Frederick, Maryland, March 23, 2017.

31. "The Science and Humanity of Chronic Traumatic Encephalopathy". Professional Development Working Group Seminar, Department of Defense, Congressionally Directed Medical Research Programs, Ft. Detrick, Maryland, March 22, 2017.

32. "Conformational Intelligence and the Humanity of Science", Dept of Pathology and Laboratory Medicine Grand Rounds, Emory University, School of Medicine, Department of Pathology and Laboratory Medicine, Atlanta, Georgia, March 13, 2017.

33. "A Morning with Dr. Bennet Omalu", CTE, FTD: Connecting the Dots- Focus on Tau, Frontotemporal Labor Degeneration Association, The University of Texas Health Science Center, San Antonio, Texas, March 11, 2017.

34. "A Morning with Dr. Bennet Omalu", Key Note Speaker, 2017 Breakfast with Friends, Catholic Charities Community Service, Rochester, New York, March 3, 2017.

35. "An Evening with Dr. Bennet Omalu". 2017 Sanderson Lecture, Thomas J. Long School of Pharmacy and Health Sciences, University of the Pacific, Stockton, California, March 1, 2017.

36. "Concussion + The Mustard Seed Effect- How Small Steps Can Spark Big Change". Chambers Lecture Series, Boston College, Chestnut Hill, Massachusetts, February 23, 2017.

37. "The Science and Consequences of Concussions: Shedding Light on CTE Disease", UC Davis, Capitol Speaker Series, UC Center Sacramento, 1130 K Street, LL22, Sacramento, CA 95814, February 22, 2017.

38. "A Morning with Dr. Bennet Omalu, Unlocking Your Full Potential: how to have a positive impact on communities, families and the world while navigating the complexities of wealth." The Threshold Group, Sausalito, California, February 22, 2017.

39. "Chronic Traumatic Encephalopathy, My Life and My Work". Court of Appeal, 3rd Appellate District, 914 Capitol Mall, Sacramento, CA 95814, January 5, 2017.

40. "An Evening with Dr. Bennet Omalu". Banner Bon Sante Ball, Banner Health Foundation, Scottsdale, Arizona, December 10, 2016.

41. "An Evening with Bennet Omalu". Pacific Northwest Chapter, World President's Organization and Young President's Organization. Concussions in Sports meeting, Swedish Neuroscience Institute, Seattle Washington, November 9, 2016.

42. "Don't Break the Rules, Change the Game", Missouri Hospital Association, 94th Annual Convention and Tradeshow, Osage Beach, Missouri, November 3, 2016.

43. "An Afternoon with Bennet Omalu". Better Business Bureau, Inc. Annual Integrity Awards Luncheon, La Vista, Nebraska, November 2, 2016.

44. "The Science and Humanity of Chronic Traumatic Encephalopathy". Navy SEAL Foundation, Inc. Navy SEAL Foundation Mental Health and Veteran Care Convening, San Diego, California, November 1, 2016.

45. "An Afternoon with Bennet Omalu". California Primary Care Association, 2016 Annual Conference, Long Beach, California, October 27, 2016.

46. "An Evening with Bennet Omalu". University Distinguished Speaker Event, Healthy Nevada Speaker Series, University of Nevada, Reno, Nevada, October 25, 2016.

47. "A Morning with Bennet Omalu". American Academy of Physical Medicine and Rehabilitation, 2016 Annual Assembly, New Orleans, Louisiana, October 21, 2016.

48. "An Afternoon with Bennet Omalu". American Health Information Management. 2016 AHIMA Annual Convention, Baltimore, Maryland, October 19, 2016.

49. "An Evening with Dr. Bennet Omalu". Key Note Speaker, El Paso Hispanic Chamber of Commerce, Dreams and Visions to Reality Equal, Annual Fiesta Celebration, El Paso, Texas, October 15, 2016.

50. "An Evening with Bennet Omalu", Patricia H. Snyder Concert and Lecture Series, University of Evansville, Evansville, Indiana, Annual Endowed Lecture and Concert Series on Innovation, research, and healthcare, October 13, 2016.

51. "A morning with Bennet Omalu". Annual Meeting, Indiana Hospital Association, Indianapolis, Indiana, October, 6, 2016.

52. "An Evening with Bennet Omalu". University Lecture Series, Miami University of Ohio, Oxford, Ohio, October 3, 2016.

53. "An Evening with Bennet Omalu". College Endowed Lecture Series, the Anna B. Mow Symposium on Comparative Religious Ethics, Bridgewater College, Bridgewater, Virginia, September 28, 2016.

54. "Don't break the rules, change the game: How Bennet Omalu single-handedly changed American Football, Professional sports, and how the world perceives traumatic brain injuries." Spotlight Program Board's Distinguished Speaker Series, Georgia State University, Atlanta, Georgia, September 27, 2016.

55. "Don't Break the Rules, Change the Game". Key note speaker, The Brain Injury Association of Michigan, 36th Annual Fall Conference, Lansing, Michigan, September 22, 2016.

56. "An Evening with Bennet Omalu", "Lost and Found" – Thrival Innovation Day 1, Thrival Festival, Thrill Mill, Inc, Pittsburgh, Pennsylvania, September 20, 2016.

57. "The Humanity of Science and Conformational Intelligence: The Bennet Omalu Story", Westminster College Hancock Symposium, The Green Lecture, Westminster College, Fulton, Missouri, September 15, 2016.

58. "An Evening with Bennet Omalu". University of Missouri, Columbia, Annual Delta Gamma Lecture Series on Values & Ethics, Jesse Auditorium, September 14, 2016.

59. "An Afternoon with Bennet Omalu". KPMG's International Partners Meeting, Prague, Czech Republic, September 12, 2016.

60. "An Evening with Bennet Omalu: Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American football, Professional sports, and how the world perceives traumatic brain injuries. University of Tennessee, Alumni Memorial Building, Cox Auditorium, Knoxville, Tennessee, August 31, 2016.

61. "An Afternoon with Bennet Omalu" Leadership Tyler. Leadership Live 2016, Tyler, Texas. August 30, 2016.

62. "A Forensic Pathologist, Chronic Traumatic Encephalopathy [CTE] and Conformational Intelligence [CI]". Keynote Speaker, American College of Legal Medicine, 22nd Annual World Congress of Medical Law, Los Angeles, California, August 10, 2016.

63. "Chronic Traumatic Encephalopathy: A Journey from Pathology Discovery to Advocacy and Beyond." Association of Pathology Chairs, 2016 Annual Meeting, San Diego, California, July 15, 2016.

64. "Don't Break the Rules, Change the Game". Keynote Address, Society of Corporate Secretaries and Governance Professionals, Annual National Conference, Colorado Springs, Colorado, June 25, 2016.

65. "Working as a Team to Seek the Truth: Bennet Omalu's Journey on Discovering CTE and Making the Movie Concussion". Key note speaker, Association of Nigerian Physicians in the

Americas [ANPA], 2016 Convention and Scientific Assembly, Las Vegas, Nevada, June 23, 2016.

66. "Interview with Dr. Omalu, moderated by Buzz Bissinger". Aspen Ideas Festival Spotlight Health, The Aspen Institute, Aspen, Colorado, June 24, 2016.

67. "Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American football, Professional sports, and how the world perceives traumatic brain injuries." Vizient New England CEO Executive Board Meeting, York, Maine. June 22, 2016.

68. "Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American football, Professional sports, and how the world perceives traumatic brain injuries" Vizient Upper Midwest Executive Board Meeting, Chicago, Illinois, June 14, 2015.

69. "Interview with Will Smith and Bennet Omalu, moderated by Jim Greenwood, CEO, BIO [Biotechnology Innovation Organization]", 2015 BIO International Convention, San Francisco, California, June 7, 2015.

70. "Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American football, Professional Sports, and how the world perceives traumatic brain injuries". Vizient New England CEO Executive Board Meeting, Williamsburgh, Virginia, June 2, 2016.

71. 2016 Project Play Summit, The Aspen Institute, Newseum, Washington, DC, May 17, 2016.

72. A Moring with Dr. Bennet Omalu. Western Health Care Leadership Academy, California Medical Association, San Francisco, California, May 15, 2016.

73. An Evening with Dr. Bennet Omalu. Drexel Neurosciences Institute Inaugural Neurosciences Conference. Drexel Neuroscience Institute, Drexel University College of Medicine, Atlantic City, New Jersey, May 11, 2016.

74. A Morning with Bennet Omalu. US Trust President's Award Program, Bank of America/ U.S. Trust, Colorado Springs, Colorado, May 10, 2016.

75. An Afternoon with Bennet Omalu. Annual SAS Health Care and Life Sciences Executive Conference, SAS Institute, Inc., Cary, North Carolina, May 4, 2016.

76. An Afternoon with Bennet Omalu. 2016 Diversity & Inclusion Symposium, Carolinas Healthcare System, Charlotte, North Carolina, April 28, 2016.

77. An Evening with Bennet Omalu. Healthcare Business Summit, MedAssets, Las Vegas, Nevada, April 26, 2016.

78. An Evening with Bennet Omalu. The Institute For Rehabilitation and Research [TIRR], Houston, Texas, April 21, 2016.

79. The 2016 Significant Speaker Event, Gallogly Events Center, University of Colorado, Colorado Springs. An Evening with Dr. Bennet Omalu: Don't Break the Rules, Change the Game: How Bennet Omalu single-handedly changed American football, professional sports, how the world perceived traumatic brain injuries, and making small steps to spark a major change. April 19, 2016.

80. Speaker, W.L. Mellon Series, Tepper School of Business, Carnegie Mellon University, April 14, 2016. The W.L. Mellon Speaker Series enables students to interact with global leaders, CEOs and management experts in student forums that encourage insightful and lively dialogue.

81. An Evening with Dr. Bennet Omalu, University of Pécs Medical School, Pécs, Hungary, April 1, 2016.

82. "A Morning with Dr. Bennet Omalu". The Head Injury Association: Head Injury Awareness Forum...Protecting School-aged Athletes from Concussion. Hauppauge, New York, March 30, 2016.

83. "Concussion: The Story Behind the Movie". 68th Annual Meeting, San Francisco Neurological Society, Sonoma, California, March 12, 2016.

84. "Don't Break the Rules, Change the Game". 11th Annual Brain Injury and Rehabilitation Conference, Liberty Station Conference Center, The Rehabilitation Center at Scripps Memorial Hospital Encinitas, San Diego, California. March 11, 2016.

85. "An Evening with Bennet Omalu". Oklahoma State University, Speakers' Board, Wes Watkins Center, Stillwater, Oklahoma, March 9, 2016.

86. "An Afternoon with Bennet Omalu". Vivo Capital Annual Limited Partner Meeting. Palo Alto, California, March 2, 2016.

87. "An Afternoon with Bennet Omalu- Discussing the Discovery of CTE". Wounded Warrior Battalion (West), Camp Pendleton, California, March 1, 2016.

88. "An Afternoon with Bennet Omalu". California Schools Employee Association and California Schools VEBA- Benefit University. Anaheim, California, February 29, 2016.

89. "An Evening with Bennet Omalu". Inspiring Minds Speaker Series, Beth El Synagogue, St. Louis Park, Minnesota, February 25, 2016.

90. "An Afternoon with Bennet Omalu". California Schools VEBA/ United HealthCare, Sacramento, California, February 23, 2016.

91. "Concussion: An Evening with Bennet Omalu". Ohio State University, Columbus, Ohio, February 18, 2016.

92. "An Evening with Bennet Omalu". Lifelong Learning Society, Florida Atlantic University, Jupiter, Florida, February 16, 2016.

93. "A Morning with Bennet Omalu". 2016 MedAssets Healthcare Executive Forum. The Westin Kierland Resort & Spa, Scottsdale, Arizona, February 6, 2016.

94. "50 Minute Interview with Stone Phillips". City Arts & Lectures, Nourse Theatre, San Francisco, California, February 4, 2016.

95. "An Evening with Bennet Omalu". Frontier Forum Lecture Series, Straz Center for the Performing Arts, University of South Florida, Tampa, Florida, December 3, 2015.

96. Keynote speaker: "Courageous Truth-Seeker: One Pathologist's Quest for a Diagnostic Breakthrough". The College of American Pathologists Annual Conference [CAP] 2015, Nashville, Tennessee, October 4, 2015.

97. Panelist, Concussion Litigation: Past, Present and Future. California Judges Association, 2015 Mid-Year Conference. May 1-3, 2015, Indian Wells, California.

98. Keynote speaker, Law Day Luncheon 2015. The San Joaquin County Bar Association and Foundation. Thursday April 30, 2015.

99. Chronic Traumatic Encephalopathy [CTE] and Emerging In-Vivo Diagnosis. Santa Clara Valley Brain Injury Conference. Conversations on Coma to Community. February 5-7, 2015, Santa Clara Marriott, Santa Clara, California.

100. The Historical Foundation of Chronic Traumatic Encephalopathy [CTE]: The Role of the Medical Examiner. West Coast Training Conference "Games People Play". Wednesday, April 23, 2014. County of Los Angeles Department of Medical Examiner-Coroner. Beverly Garland, North Hollywood, Los Angeles, California.

101. Emerging In-Vivo Diagnosis of Chronic Traumatic Encephalopathy. Grand Rounds Lecture, Monday, December 2, 2013. Department of Medical Pathology and Laboratory Medicine, University of California, Davis.

102. Chronic Traumatic Encephalopathy: Current Understanding of Clinicopathologic Features; A Forensic Perspective. American Academy of Neurology, 65th Annual Meeting. San Diego, March 16-23, 2013. San Diego Convention Center. March 21, 2013: Cumulative Sports Concussion and Risk of Dementia Course.

103. CTE: A Historical Perspective- Pathological Overview and Taupathy. Mild Head Injury, Concussion, and Return to Activities: Update 2013. Symposium, Friday, January 25, 2013. The University of Chicago, Section of Neurosurgery, Department of Surgery. The University of Chicago Gleacher Center, 450 North Cityfront Plaza Drive. Chicago, Illinois.

104. History, Definition and Clinicopathologic Features of Chronic Traumatic Encephalopathy [CTE]. Sports Concussion Institute, The National Summit On Sports Concussion And Other Athletic Injuries. June 22, 2012. Los Angeles, California.

105. "Pathophysiology of Traumatic Brain Injury". Manteca Unified School District, School Nurses. Manteca, California. March 19, 2012.

106. "Sociology of Health & Illness". Department of Sociology, University of Pacific, Stockton Campus, Stockton, California. March 15, 2012.

107. "Chronic Traumatic Encephalopathy and PTSD". Grand Rounds, Department of Neurosurgery, University of California, Davis, Sacramento, California, March 13, 2012.

108. "Motivational Lecture and Forensics: Becoming What You Want to Become" San Joaquin County Youth Leadership Academy, District Attorney's Office, June 27, 2012.

109. "Youth Leadership for Change", 2012 Law Day Educational Program, San Joaquin County Bar Association. April 26, 2012.

110. "Chronic Traumatic Encephalopathy and PTSD" Full-Day Dementia Program, San Francisco Neurological Society. Sir Francis Drake Hotel, San Francisco, California, Friday, October 14, 2011.

111. "Taupathy and Chronic Traumatic Encephalopathy" Grand Rounds, Department of Pathology, University of California-Davis, Sacramento, California. April 18, 2011.

112. "Long Term Effects of Repeated Impacts to the Head in American Athletes". Brain Rehabilitation and Injury Network. Thinktank 2011, Long Beach, California. March 27, 2011.

113. "Chronic Traumatic Encephalopathy in American Athletes". Visiting Professor, Grand Rounds, University of California, San Francisco, Department of Neurology, Memory and Aging Center, March 11, 2011.

114. Motivational Lecture to One Hope School, County Operated Schools and Programs, San Joaquin County Office of Education. October 24 and 25, 2011

115. "Motivational Lecture and Forensics: the value and importance of education" San Joaquin County Youth Leadership Academy, District Attorney's Office, June 29, 2011.

116. Employee Personal Motivation Lecture. Employee Mentoring Program, Equal Employment Opportunity Office, San Joaquin County, Administration Building, 44 North San Joaquin Street, June 16, 2011.

117. Keynote Speaker, Thirteenth Annual San Joaquin County Diversity Luncheon. San Joaquin County, California. September 30, 2010.

118. "Becoming a Forensic Pathologist: My Experience and Perspective". Guest Lecturer, First and Second Year Medical Students, School of Medicine, University of California, Davis, October 22, 2010.

119. "Chronic Traumatic Encephalopathy in Sports". 2010 Advanced Team Physician Course, American College of Sports Medicine, American Medical Society for Sports Medicine and American Orthopaedic Society for Sports Medicine. Washington, DC, December 10, 2010.

120. "Emerging Technology in the Evaluation and Treatment of Concussion". 2010 Advanced Team Physician Course, American College of Sports Medicine, American Medical Society for Sports Medicine and American Orthopaedic Society for Sports Medicine. Washington, DC, December 10, 2010.

121. "Epidural Hemorrhages" Visiting Professor Lecture, Neurosurgery Residents, Department of Neurosurgery, West Virginia University, August 17, 2010

122. "Pharm Parties". 10th Annual San Joaquin County Child Abuse Prevention Symposium, Stockton, California, April 14, 2010.

123. "The Forensic Pathology of Chronic Traumatic Encephalopathy". 2010 Independent Retired Players Summit and Conference, Las Vegas, Nevada, April 17, 2010.

124. "Chronic Traumatic Encephalopathy". The Amen Clinic, Newport Beach, California, March 20, 2010.

125. "The forensic pathology of Chronic Traumatic Encephalopathy in American Athletes: the journey thus far." Advanced Education Seminar, Lakewood Orthopedics & Sports Medicine, Speakers Bureau Member, Dallas, Texas, January 23, 2010.

126. "Emerging Forensic Issues of Chronic Traumatic Encephalopathy in American Athletes". Presenter, 2009 National Educational Conference, American Association of Legal Nurse Consultants, Phoenix, Arizona, April 24, 2009.

127. "The stressors in the life of a physician and forensic pathologist". Sociology of Health & Illness Course, University of the Pacific, Stockton, California, March 26, 2009.

128. "New Technologies in Neuropathology and Neuropathology of Concussion in Athletes". Presenter, Emerging Technology and Techniques in Neurosurgery, Ceasar's Palace, Las Vegas, November 8, 2008, Departments of Neurosurgery, West Virginia University, Morgantown, WV, and Allegheny General Hospital, Pittsburgh, PA.

129. "The Forensic Pathology of Chronic Traumatic Encephalopathy in Professional American Athletes". Key Note Speaker, American Association of Legal Nurse Consultants, West Virginia, Upper Ohio Valley Chapter, 2008 meeting, October 17, 2008, Wheeling, West Virginia.

130. "The Pathophysiology of Traumatic Brain Injury". Visiting Professor/Guest Lecturer, October 1, 2008, Department of Neurosurgery, West Virginia University, Morgantown, West Virginia.

131. Key Note Speaker, "Helping People One Test at a Time", National Laboratory Week, Harlem Hospital Center, New York City, April 25, 2008

132. "The Link between the Field and Dementia" Rendezvous II: An International Sports Medicine Conference, March 25, 2008, Caesars Palace, Las Vegas. 17th Annual Meeting of The American Medical Society for Sports Medicine (AMSSM) and The Canadian Academy of Sports Medicine (CASM), March 25 – 29, 2008.

133. "The Neuropathology and Delayed Sequelae of Concussion in NFL Players". The National Concussion Summit: Concussion in Sports: The Under-Recognized Public Epidemic The Sports Concussion Institute. Centinela Freeman Regional Medical Center, Marina Campus, Marina Del Rey, California at The Marriott Hotel, 4100 Admiralty Way, Marin Del Rey, CA 90292, Friday, April 20th, 2007.

134. "Football Induced Chronic Traumatic Encephalopathy" November 2006, meeting of the Pittsburgh chapter of the National Association of Neurological Nurses, Waterfront, Homestead, Pittsburgh.

135. "The forensic perspectives of traumatic brain injury". 2005 Annual Nursing Conference: traumatic brain injury, from ER management to ICU, rehab, organ donation and Trisha Meili a.k.a. Central Park Jogger. Allegheny General Hospital Pittsburgh, Pennsylvania. November 18, 2005

136. 'Mortality following bariatric surgery'. Bariatric Surgery Research Mini-symposium, Minimally Invasive Surgery Center, Department of Surgery, University of Pittsburgh, Pennsylvania, January 17, 2004.

137. "My professional life as a forensic pathologist and neuropathologist". A career development presentation to selected high school students of the Pittsburgh Public School System. April 14, 2004.

138. "My professional life as a forensic pathologist and neuropathologist". A career development presentation to students of the Saint Benedict the Moor Catholic School, 2900 Bedford Avenue, Pittsburgh, Pennsylvania 15219. March 5, 2004.

139. 'The medico-legal autopsy'. University of Pittsburgh Chapter of the American Inns of Court, Pittsburgh, Pennsylvania. April, 2002.

140. 'My professional experience as a Forensic Pathologist'. Graduating Lecture, 2002 certificate program class, The Cyril H. Wecht Institute of Forensic Sciences, School of Law, Duquesne University, Pittsburgh, Pennsylvania. November, 2002.

141. "The mechanisms of trauma in motor vehicular accidents". Surgical grand-rounds, Department of Surgery, The Mercy Hospital System, Pittsburgh, Pennsylvania. May, 2000.

Post-Graduate Medical Teaching Experience-1

1. Supervised medical students during the Emergency Medicine rotation, University of Jos, Jos, Nigeria, January 1991-September, 1994.

2. Taught a patho-physiology course for the physician assistant program at the Harlem Hospital Center, New York, NY. July, 1996 – June, 1999.

3. Presented weekly clinico-pathologic conferences for the departments of pathology, surgery, internal medicine, obstetrics and gynecology, Harlem Hospital Center, New York, NY. July, 1995 – June, 1999.

4. Taught medical students and residents, forensic pathology and neuropathology, University of Pittsburgh, July, 1999 – June, 2002.
5. Taught a workshop in the patho-physiology course, Masters in Public Health program, Graduate School of Public Health, University of Pittsburgh, September, 2002 – 2007.

6. Supervised residents during rotation in forensic pathology, University of Pittsburgh, July, 2002 – 2007.

7. Taught monthly forensic neuropathology workshops to residents and fellows in pathology, neurology and neurosurgery, University of Pittsburgh, 2004 – 2007.

8. Taught monthly neuropathology workshops to residents in pathology, Conemaugh Memorial Hospital, Johnstown, Pennsylvania, January 2005 – 2007.

9. Presented monthly trauma rounds to surgery residents, Mercy Hospital of Pittsburgh, January 2006 – 2007.

10. Presented quarterly one-day introductory autopsy conferences for the emergency medicine technology program of Allegheny County Community College, April, 2006.

11. Forensic pathology and neuropathology grand round lectures, internal medicine, family practice and surgery residents, San Joaquin General Hospital, French Camp, California, 2009 – 2012.

12. Forensic neuropathology brain cutting workshops for pathology residents and fellows, University of California at Davis, Department of Pathology, 2009 – 2013.

13. Forensic neuropathology brain cutting workshops for neurology residents and fellows, University of Nevada, Las Vegas at the Clark County Coroner's Office, 2011 – 2012.

14. Mentoring and teaching residents and medical Students and covering the autopsy service, and forensic neuropathology service, Department of Medical Pathology and Laboratory Medicine, University of California at Davis, March 2012 – July 2013.

15. Microscopy sessions and slides review, residents and medical students, Department of Medical Pathology and Laboratory Medicine, University of California at Davis, September 2013 to present.

16. Grand rounds lectures, didactic lectures in laboratory management, forensic pathology and neuropathology for medical students, residents and fellows, Department of Medical Pathology and Laboratory Medicine, UC-Davis Medical School, 2008 – Present.

17. Grand Rounds, Department of Medical Pathology and Laboratory Medicine, UC-Davis, February 1, 2016: What a Pathologist can Learn from Hollywood: Dr. Omalu's Experience.

18. Brain Cutting Workshops and Signouts, Medical Students, Graduate Medical and Non-Medical Students, Residents and Fellows, Department of Medical Pathology and Laboratory Medicine, University of California- Davis Medical School. January 2018 – Present.

Research Grant

1. $24, 750.00 grant from The Pittsburgh Foundation for a research project on Loss of heterozygosity as predictors of grade and outcome in meningiomas in collaboration with John Lee, M.D. University of Pittsburgh.

2. $200,000.00 grant from The Hazel Ruby Mcquain Charitable Trust, West Virginia to the Brain Injury Research Institute, for research into Chronic Traumatic Encephalopathy, 2010-2011.

Boards and Committees Memberships

1. Child Death Review Board, San Joaquin County, California, 2007 – 2018

2. Infant Death Review Board, San Joaquin County, California, 2007 – 2018

3. Elder Death Review Board, San Joaquin County, California, 2007 – 2018

4. Homicide Review Committee, San Joaquin County, California, 2010 – 2015

5. San Joaquin County Trauma Audit Committee, San Joaquin County Emergency Medical Services Agency, California, 2014 – 2018

6. Member, Traumatic Brain Injury Advisory Board, State of California, 2018 - Present

Broadway Theatre and Hollywood Motion Picture Coverage

1. Concussion, drama film, Sony Pictures, starring Will Smith as Bennet Omalu. December 25, 2015. Written and directed by Peter Landsman, produced by Scott Free Productions and the Shuman Company.

2. League of Denial: The NFL's Concussion Crisis. Documentary Movie. Frontline, Public Broadcasting Service [PBS]. October 8, 2013. http://www.pbs.org/wgbh/frontline/film/league-of-denial/?utm_source=email&utm_medium=social&utm_campaign=share_button

3. EST/Sloan presents Headstrong, by Patrick Link; directed by William Carden. The Ensemble Studio Theatre, New York City, New York. April/May 2012.

Selected Book Coverage

1. Concussion by Jeanne Marie Laskas. Random House, New York, USA. 2015 [New York Times Best Seller]

2. League of Denial. The NFL, Concussions, and the Battle for Truth by Mark Fainaru-Wada and Steve Fainaru. Crown-Archetype, Random House, New York, USA. 2013.

3. The Death Penalty on Trial, Crisis in American Justice, by Bill Kurtis, PublicAffairs™, 2004.

Selected Media Coverage

1. The Lost Lincoln, History Will Change. Discovery Channel TV, October 4, 2020. https://www.discovery.com/shows/the-lost-lincoln

2. Medical Examiners under Fire. CBS Sunday Morning, CBS News, June 14, 2020.

3. The Truth Seeker. Sitting Down With Bennet Omalu, President and Medical Director of Bennet Omalu Pathology, by Luke Turner, The Pathologist, September 27, 2019. https://thepathologist.com/outside-the-lab/the-truth-seeker

4. Contact sports will "cease to exist" within a generation. Adrian Proszenko. The Sidney Morning Herald. August 11, 2019. https://www.smh.com.au/sport/nrl/contact-sports-will-cease-to-exist-within-a-generation-20190809-p52fpf.html

5. Renowned Pathologist Testifies in Football Player's Lawsuit. Barbara S. Miller. The Intelligencer: Wheeling News Register. May 12, 2019. http://www.theintelligencer.net/news/community/2019/05/renowned-pathologist-testifies-in-football-players-lawuit/

6. Recognizing the patterns of truth. You may have seen the movie based on the life of Bennet I. Omalu, MD, MBA, MPH. Find out the real story behind what he has discovered in CTE and in letting science lead the way. By Kevin O'Reilly. American Medical Association Moving Medicine Magazine. Spring 2019, Volume 1, Issue 1. March 30, 2019. https://app.svwps.com/americanmedicalassociation/ama/amamag/mag.html

7. Bennet Omalu, le médecin qui a secoué le sport En faisant le lien au début des années 2000 entre dégénérescence cérébrale et pratique du football américain, ce médecin légiste a brisé un tabou. Il s'est attiré les foudres de la NFL mais ses travaux ont fait évoluer les mentalités et eu des répercussions en hockey, rugby et football. Valérie de Graffenried, Le Temps, February 15, 2019. https://www.letemps.ch/sport/bennet-omalu-medecin-secoue-sport

8. Was C.T.E. Stealing His Mind? A Gunshot Provided the Answer: Jason Hairston played briefly in the N.F.L., hunted with Donald Trump Jr. and owned a wildly successful hunting gear and apparel company. But he increasingly worried about a degenerative brain disease. John Branch, New York Times, January 31, 2019. https://www.nytimes.com/2019/01/31/sports/cte-football-hairston-super-bowl.html

9. REVEALED: Former NFL star Jason Hairston was home alone with his son, 10, when he took his life - moments after the boy said he was 'acting different' in a call to his mom, who says CTE is to blame. Chris Spargo, Daily Mail, February 2019. https://www.dailymail.co.uk/news/article-6155381/NFL-star-Jason-Hairston-home-son-10-suicide-autopsy-looks-CTE.html

10. Tout ce que je cherchais, c'est la vérité - Dr Bennet Omalu. Propos recueillis par Diane Sauvé, Journaliste, Radio-Canada Sports. January 8, 2019. https://ici.radio-canada.ca/sports/podium/68/podium-dr-bennet-omalu-commotions-concussion-cerebrales-football-will-smith-cte-etc
http://ici.radio-canada.ca/sports/podium/70/podium-connor-crisp-hockey-commotions-cerebrales-omalu-canadien-etc-cte?fromApp=appSportsAndroid&fromMobileApp=android

11. The Forensic Fighter: From Battling the NFL to challenging police shootings, Dr. Bennet Omalu has used his scalpel to expose the truth. By Gabriel Thompson, San Francisco Magazine, October 19, 2018. https://modernluxury.com/san-francisco/story/the-forensic-fighter

12. Head Drama- When Bennet Omalu, MD, identified a degenerative brain disease in NFL players, it shifted the worlds of both sports and neurology- and protocols about concussion. By Richard Laliberte, Brain&Life, Neurology For Everyday Living. American Academy of Neurology, August/September 2018.

13. Independent Autopsy Finds Police Shot Stephon Clark in the Back. Julie Small, KQED News, March 30, 2018. https://www.kqed.org/news/11658808

14. Favre and famed doctor on the NFL's concussion crisis. https://www.cnn.com/videos/tv/2018/02/01/bennet-omalu-brett-favre-amanpour.cnn/video/playlists/amanpour/ Christiane Amanpour, February 1, 2018.

15. CTE Has Been Found in a Living Brain for the First Time Ever: Dr. Bennet Omalu has made a big breakthrough. By Luke Darby, GQ magazine, November 17, ,2017. https://www.gq.com/story/cte-living-brain

16. Ex-NFL player confirmed as 1st case of CTE in living patient. By Nadia Kounang. CNN Health, November 16, 2017. http://www.cnn.com/2017/11/16/health/cte-confirmed-in-first-living-person-bn/index.html

17. Ex-NFL player confirmed as first case of CTE found in living person – researchers. By Bryan Armen Graham. The Guardian, November 16, 2017. https://www.theguardian.com/sport/2017/nov/16/cte-living-person-nfl-concussion-brain-trauma-research

18. Sacramento's Dr. Bennet Omalu and His Struggle for Sports Safety. By Seth Sandronsky. Comstock's Business Insight for the Capital Region, November 9, 2017. https://www.comstocksmag.com/web-only/sacramentos-dr-bennet-omalu-and-his-struggle-sports-safety

19. Book by 'Concussion' doctor is recommended reading for parents of football kids. By Ed Sherman Chicago Tribune, October 30, 2017. http://www.chicagotribune.com/lifestyles/books/sc-books-sports-book-roundup-1101-story.html

20. "From Hopeless to Healer: Persecuted in Nigeria, Stonewalled by the NFL- Early struggles prepared Bennet Omalu for character assassination in the U.S. By **Error! Hyperlink reference not valid.**

21. 'Concussion' doctor says kids shouldn't play these sports until they're 18. By A. Pawlowski. Health and Wellness, September 5, 2017. https://www.today.com/health/concussion-doctor-warns-against-contact-sports-kids-t115938

22. "Truth Doesn't Have a Side". Publishers Weekly, Book Review, August 2017. https://www.publishersweekly.com/978-0-310-35196-2?utm_source=Publishers+Weekly&utm_campaign=92aa48e849-EMAIL_CAMPAIGN_2017_08_23&utm_medium=email&utm_term=0_0bb2959cbb-92aa48e849-305322873

23. "Who is Bennet Omalu and why did his concussion discovery send shock waves through sport?", by Jeremy Wilson, The Telegraph, August 9, 2017. http://www.telegraph.co.uk/football/2017/08/09/bennet-omalu-did-concussion-discovery-send-shock-waves-sport/

24. New book shows a more intimate 'Side' of Dr. Bennet Omalu, by Terri Schlichenmeyer, The Philadelphia Tribune, August 22, 2017. http://www.phillytrib.com/lifestyle/new-book-shows-a-more-intimate-side-of-dr-bennet/article_e969b814-adac-5897-ac64-1eb5cc56166a.html

25. 'Concussion' doctor: No such thing as 'making football safer', by Brett Cyrgalis, New York Post, August 7, 2017. http://nypost.com/2017/08/07/concussion-doctor-no-such-thing-as-making-football-safer/

26. Dr. Bennet Omalu: CTE obsession obscuring truth about brain health of football players, by Kevin Seifert, ESPN, August 4, 2017. http://www.espn.com/nfl/story/_/id/20245394/dr-bennet-omalu-says-obsession-cte-obscuring-larger-truth-brain-health-football-players

27. Under 18s should not play rugby, says 'concussion' doctor. Leading neuropathologist calls for contact sport with risk of head blows to be limited to adults, by Gavin Cummiskey, The Irish Times, Dublin, Ireland, June 3, 2017. http://www.irishtimes.com/sport/rugby/under-18s-should-not-play-rugby-says-concussion-doctor-1.3105970

28. How head injuries will end sport as we know it. Dr Bennet Omalu says children should not be playing rugby or heading the ball in soccer, by Gavin Cummiskey, The Irish Times, Dublin, Ireland, June 3, 2017. http://www.irishtimes.com/sport/rugby/how-head-injuries-will-end-sport-as-we-know-it-1.3105943

29. http://www.post-gazette.com/local/city/2017/05/30/Bennet-Omalu-concussion-Kenneth-Reeves-homicide-trial-Allegheny-County-Pittsburgh/stories/201705300124.

30. Omalu Says NHL Docs Using 'Alternative Facts' To Refute CTE, by Zachary Zagger, Law360, New York, February 8, 2017.

31. Bennet Omalu gets involved in NHL concussion lawsuit, by Rick Westhead, TSN Canada, February 9, 2017. http://www.tsn.ca/bennet-omalu-gets-involved-in-nhl-concussion-lawsuit-1.667427

32. Thank you Dr. Omalu, by Roger Braden, Attorney at Law Magazine, Kentucky Edition, Volume 4, No. 1, 2016.

33. Physician who discovered CTE in NFL players gets AMA's highest honor, by Timothy Smith, AMA Wire, November 12, 2016. https://wire.ama-assn.org/ama-news/physician-who-discovered-cte-nfl-players-gets-ama-s-highest-honor

34. First case of CTE diagnosed in MMA fighter, by Bob Hohler, The Boston Globe, October 21, 2016. https://www.bostonglobe.com/sports/2016/10/20/first-case-cte-diagnosed-mma-fighter/SfUZnoh3Z1dT9xruTm95RO/story.html

35. Bennet Omalu - From Trauma to Triumph, Dr. Benjamin Ola. Akande, President, Westminster College, Missouri. Ladue News, St. Louis, Missouri.

36. Bennet Omalu, MD. The Physician Leader Whose Research Inspired The Movie Concussion. PIJ- Physician Leadership Journal, Bill Steiger, American Association For Physician Leadership. Volume 3, Issue 2, March/April, 2016.

37. Truth and Consequences. Hillary Louise Johnson. Sactown Magazine. April-May 2016.

38. Concussion, Bang to Rights: Science is taking big stems toward understanding the impact of concussion. The Economist, March 5, 2016. http://www.economist.com/news/science-and-technology/21693906-science-taking-big-steps-toward-understanding-impact-concussion-bang

39. Bennet Omalu receives congressional honors, by The Record. The Stockton Record Newspaper, February 5, 2016. http://m.recordnet.com/article/20160205/NEWS/160209826.

40. CTE in the NFL: The tragedy of Fred McNeill. By Nadia Kounang, CNN. February 5, 2016. http://edition.cnn.com/2016/02/04/health/fred-mcneill-cte-football-player/index.html.

41. Brain Trust. Will Smith shines a light on the dark side of the sport he loves. By Ben Reiter. Sports Illustrated Magazine. December 28, 2015.

42. The Inside Story of Concussion's Dr. Bennet Omalu and His 'Good Friend' Will Smith. By Johnny Dodd. People Magazine. December 24, 2015.

43. The Doctor the NFL Tried to Silence. League physicians sought to discredit Bennet Omalu's autopsy study showing widespread brain damage in former Steelers star Mike Webster. By Jeanne Marie Laskas. World Street Journal. November 24, 2015.

44. Greater Good. Health Heroes- Scientist. WebMD. November-December 2015 issue.

45. Concussion Pathologist Play by Will Smith Tells His Story. Pam Harrison. Medscape Medical News. Medscpae.com. October 4, 2015.

46. Gridiron Dementia. Nicholas Ducassi. Carnegie Mellon Today. May 2015 issue.

47. Will Smith to play Bennet Omalu, who changed the way we think about football. Alyssa Rosenberg. The Washington Post, June 5, 2014 [http://www.washingtonpost.com/news/act-four/wp/2014/06/05/will-smith-to-play-bennet-omalu-who-changed-the-way-we-think-about-football/]

48. Will Smith to Star In NFL Concussion Drama (EXCLUSIVE): Sony's untitled thriller based on GQ article 'Game Brain'. Justin Kroll. Variety, June 3, 2014. [http://variety.com/2014/film/news/will-smith-to-star-in-movie-about-nfl-concussions-exclusive-1201210878/]

49. Dorsett, others show signs of CTE. By William Weinbaum and Steve Delsohn | ESPN.com, November 6, 2013. [http://espn.go.com/espn/otl/story/_/id/9931754/former-nfl-stars-tony-dorsett-leonard-marshall-joe-delameilleure-show-indicators-cte-resulting-football-concussions].

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52. ESPN.com, January 22, 2013. "CTE found in living ex-NFL players" by Steve Fainaru *and* Mark Fainaru-Wada

53. The New York Times, April 26, 2012. "Veterans and Brain Disease" by Nicholas D. Kristof.

54. Gehirn&Geist, February 9, 2012, Bidredaktion, SCHÄDEL-HIRN-TRAUMATA, Krieg im Kopf [German]; by Sharon Weinberger

55. "Gun linked to Gilley in killing" by Jennie Rodriguez-Moore. The Stockton Record. April 17, 2012.

56. Nature 477**,** 390-393 (September 2011) Bombs' hidden impact: The brain war. Wartime explosions may be creating an epidemic of brain damage — and a major challenge for scientists; by Sharon Weinberger.

57. Faculty Newsletter, UC Davis Health System, February – March 2011: Brain Trauma Expert Bennet Omalu Teaches Forensic Neuropathology; Published by the Faculty Development Office.

58. ESPN The Magazine, January 10, 2011: Coming to a head: When the NFL reversed field on concussions, it spawned a medical gold rush that pits scientist against scientist in a quest for cures, treatments and dead men's brains; by Peter Keating.

59. The Patriot-News, December 26, 2010: 2010: The year of the concussion; by Stefanie Loh.

60. CNN.com, November 24, 2010: Ex-NFL stars after concussion: Lives unraveled; by Stephanie Smith, CNN medical producer.

61. CNN.com, November 22, 2010, Warner: Playing through concussion 'part of the game'; by Stephanie Smith, CNN Medical News.

62. The Patriot-News, August 22, 2010: What Chris Henry taught us: how an autopsy of the former Cincinnatic Bengals' receiver's brain has helped doctors further research about concussions in football, by Stefanie Loh.

63. The New York Times, June 28, 2010: Former Bengal Henry Found to Have Had Brain Damage, by Alan Schwarz.

64. "Gray jury report unveils details of Huckaby case" by Scott Smith. The Stockton Record. June 19, 2010.

65. The Associated Press, June 18, 2010: Forensic Pathologist: Disposal of Sandra Cantu's Body Likely Premeditated.

66. Fox News Network, American News HQ, Shannon Bream, Sunday, February 7, 2010.

67. ESPN Television, Outside the Lines, Concussions in Football, Sunday, February 7, 2010.

68. Stars and Stripes, "Doctors study link between combat and brain disease." January 23, 2010, by Seth Robbins.

69. The Sydney Morning Herald, Australia, "US football players face head injury time bomb." January 10, 2010, by Ed Pilkington.

70. Guardian Newspaper, United Kingdom, "Ticking timebomb in US colleges as American football head injuries linked to dementia: As NFL bows to pressure and changes its rules, a Congressional hearing has opened into fears of long-term brain damage." January 4, 2010, by Ed Pilkington.

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72. "S.J. medical examiner details gruesome scene", by Scott Smitt. The Stockton Record. November 20, 2009.

73. ABC Television, Nightline: Driven Mad? What long-term brain injuries can football players sustain? October 16, 2009, by Martin Bashir and Roxanna Sherwood.

74. GQ Magazine: Blowing the whistle on the NFL: a shocking look at what the modern game is doing to players' brains. October 2009, by Jeanne Marie Laskas.

75. Science Journal: A late hit for pro football players: emerging research suggests that hard knocks on the field may cause delayed brain damage in retired athletes. August 7, 2009, by Greg Miller.

76. Canadian Broadcasting Corporation- Television Documentary: Head Games. The Fifth Estate. Aired on Wednesday November 19, 2008 on CBC-TV.

77. Canadian Broadcasting Corporation-Television Documentary: A fight to the death. Concussions in Sports. Aired on February 6 and 10, 2008 on CBC-TV and on February 8 on CBC Newsworld.

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PART 2: BIBLIOGRAPHY

Books and Book Chapters

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3. Omalu, B. Chronic Traumatic Encephalopathy in Concussion, Niranjan A and Lunsford LD eds. Progress in Neurological Surgery, Vol. 28. Karger, New York, New York, 2014.

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8. Evaluation of head, neck and spinal cord for injury and disease. Omalu, BI and Hammers JL. In Medical Legal Handbook, Hammers JL, Fitzsimmons, RP eds. Juris Publishing, Inc, 2018, Huntington, New York.

Peer-Reviewed Scientific Journal Publications - 1

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Abstract Presentations and Publications - 1

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Abstract Presentations and Publications - 2

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9. Omalu B. Knowledge, attitude and practice of contraception among university students in Enugu, Nigeria. Library Archives, Department of Public Health, College of Medicine, University of Nigeria: May 1989.

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Abstract Presentations and Publications - 3

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19. Omalu BI, "Diagnosis of Alzheimer’s disease in an exhumed decomposed brain after twenty months of burial in a deep grave”. Poster Presentation at the American Association of Neuropathologists (AANP) 2004 Annual Meeting, Cleveland, Ohio, June 24-27, 2004.

Abstract Presentations and Publications - 4

20. Omalu BI, "Chronic traumatic encephalopathy and the National Football League" Poster Presentation at the American Association of Neuropathologists (AANP) 2004 Annual Meeting, Cleveland, Ohio, June 24-27, 2004.

21. Omalu BI, "Diagnosis of Alzheimer's disease in an exhumed decomposed brain after twenty months of burial in a deep grave". Poster Presentation at the National Association of Medical Examiners (NAME) 2004 Annual Meeting, Nashville, TN, September, 10-15, 2004.

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23. Ionescu DN, Naus GJ, Shakir AM, Omalu BI. Autopsy findings, manner and cause of death in nursing home deaths investigated by the Allegheny county coroner's Office, Pennsylvania: a five year retrospective review. Poster Presentation at the United States and Canadian Academy of Pathology 2005 Annual Meeting, San Antonio, Texas, February 26 – March 4, 2005.

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Abstract Presentations and Publications - 5

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27. Lindner JL, Omalu BI, Shakir AM. Nursing home deaths, which fall under the jurisdiction of the medical examiner. An eleven year retrospective review. 2006 national meeting of the National Association of Medical Examiners. October 2006, San Antonio, Texas.

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29. Omalu BI. The emerging role of forensic pathologists in identifying cases of chronic traumatic encephalopathy in NFL players. 2007 Annual meeting of the National Association of Medical Examiners, October 12 – October 17, 2007, Savannah, Georgia.

30. Lindner JL, Omalu BI. The role of environmental factors in the causation of sudden death in infants: two cases of sudden unexpected death in two unrelated infants who were cared for by the same babysitter. 2007 Annual meeting of the National Association of Medical Examiners, October 12 – October 17, 2007, Savannah, Georgia.

31. Lindner JL, Omalu BI. Forecasting as an operations management tool in a medical examiner's office. 2007 Annual meeting of the National Association of Medical Examiners, October 12 – October 17, 2007, Savannah, Georgia.

Abstract Presentations and Publications - 6

32. Bailes J, Omalu BI. Spectrum of Consequences of Sports-Related Mild Traumatic Brain Injury. 2008 annual meeting of the American Academy of Neurological Surgeons, September 8, Phoenix, Arizona.

33. Bailes JE, Omalu B: Chronic Traumatic Encephalopathy: Autopsy analysis in professional and contact sport athletes. Presented at: American Academy of Neurological Surgery 71st annual meeting, West Palm Beach, Florida, November 2009.

34. Omalu B, Chronic Traumatic Encephalopathy: My Understanding of What We Can do Moving Forward. 13th Annual Society for Brain Mapping and Therapeutics. Miami, Florida, April 9th, 2016.

35. Omalu B, CTE and FDDNP: Blast and Non-Blast Variants. 13th Annual Society for Brain Mapping and Therapeutics. Miami, Florida, April 9th, 2016.

36. Omalu B, Chronic Traumatic Encephalopathy, Conformational Intelligence and the Law. April 23, 2017. Association of Defense Trial Attorneys, 2017 Annual Meeting, April 19-23, 2017, Monterey, California.

Internet, Newspaper and Newsletter Articles and Publications

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2. It's time to stop fooling ourselves about the harm of heading a football. Daily Mail, October 28, 2019. https://www.dailymail.co.uk/news/article-7347433/Dr-Bennet-Omalu-says-contact-sports-banned-Australia.html.

3. CTE Discoverer: We Shouldn't Be Surprised That Aaron Hernandez Had CTE. Op-Ed Contribution, Fortune.com, September 26, 2017. http://fortune.com/2017/09/26/bennet-omalu-cte-aaron-hernandez/?iid=sr-link1

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